IJAE
Vol. 118, n. 2 (Supplement): 87, 2013
ITALIAN JOURNAL OF ANATOMY AND EMBRYOLOGY

Induced Biochemical osteoporosis: Effects of 1-month calcium–deprived diet on rat bone remodelling with/ without contemporary administration of PTH(1-34)

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It is known that rats fed calcium-deprived diet develop osteoporosis due to enhanced bone resorption secondary to parathyroid overactivity resulting from nutritional hypocalcemia. Therefore, rats provide a good experimental animal model for studying bone remodelling alterations during biochemical osteoporosis. This preliminary study is performed in 3 month-old Sprague Dawley male rats, divided into 4 groups (5 rats each): 1) base line, 2) normal diet for 4 weeks, 3) calcium-deprived diet for 4 weeks; 4) calcium-deprived diet for 4 weeks plus contemporary administration of PTH(1-34) 40μg/kg/day. Three labels of osteogenesis were performed at 1st , 20th and 27th day of experimental period in order to evaluate bone formation during animal treatment. Histomorphometrical analyses were performed on cortical bone of femoral diaphyses, as well as on trabecular bone of distal femoral metaphyses, both transversely sectioned. The preliminary results showed that at femur mid-diaphyseal level the diet induced a reduction of cortical bone area (even if not significant) with enlargement of the medullary canal due to endosteal resorption, while periosteal neo-deposition is similar in all groups and particularly abundant in those periosteal regions mainly devoted in answering the mechanical demands. PTH(1-34) treatment seems to reduce endosteal resorption only in those surfaces where periosteal mechanical loading are less consistent. Conversely, PTH(1-34) treatment doesn't seem to affect osteoblast activity. Moreover, in distal femoral metaphyses, diet induced osteoclast activity, with a decrease in the amount of trabecular bone volume, confirming that this architecture is mainly devoted in answering the metabolic demands. The novelty of the proposed model Is the contemporary administration of PTH(1-34) together with calcium deprived diet to evaluate induced-biochemical osteoporosis. This model seems a good starting point for successive studies in order to study bone alterations during unbalanced calcium metabolism frequently occurring in aging and to define time and manner of bone mass recovery.

Key words

Bone remodelling, biochemical osteoporosis, calcium-deficient diet, PTH(1-34), rats.