INTRODUCTION

Osteoporosis is recognized as one of the negative factors for implant fixation between bone and cementless implant. Osteoconductivity of hydroxyapatite (HA) coatings enhances implant fixation. HA-coatings therefore is also being widely used for clinical applications. However, the previous experimentally studies have shown that the osteoconductive activity effect of HA-coated implants on implant fixation was lower in ovariectomized osteopenic rats than in healthy rats (1, 2).

Bisphosphonates and vitamin D are used for treatment of osteoporosis in human. Alendronate, a third-generation bisphosphonate, is well known as a potent inhibitor of osteoclastic bone resorption. Alendronate has also been reported to increase the bone mineral density in estrogen-deficient animals. Therefore, the inhibition agent of bone resorption may increase initial fixation between bone and implant in osteoporosis. There are also evidences that vitamin D (1,25(OH)2D3) increases bone mineral density in postmenopausal osteoporosis. However, other studies have observed negative results. The effect of vitamin D on the treatment of osteoporosis is still controversial.

The purpose of this study was to investigate the effect of separate and combined treatment with alendronate and 1,25(OH)2D3, on bone mass and initial fixation of Titanium (Ti) or HA-coated implants in ovariectomized rats.

MATERIALS AND METHODS

Forty female Wistar rats, 12 weeks of age, were divided into two, bilaterally ovariectomized (OVX: 32 rats) and control sham-operated rats (Control: 8 rats). Twenty-eight weeks later, femora of all rats were implanted intramedullary with either Ti or HA-coated implants (23 mm length and 1.4 mm diameter). The average surface roughness was 4.3 μm in Ti implants and 4.3 μm in HA-coated implants. After implantation, OVX rats were further divided into four groups (n=8/group). OVX rats were infused continuously in daily doses of saline alone or 0.1 mg/kg of alendronate or 0.1 μg/kg of 1,25(OH)2D3 or a combined treatment with alendronate and 1,25(OH)2D3 by subcutaneously implanted mini-osmotic pumps (Alzet Pump, Alza Corporation, Palo Alto, CA) for 4 weeks. The control group also received saline alone by mini-osmotic pumps for 4 weeks. At the end of this period, all animals were sacrificed and their femora and tibiae were excised.

The cross-sections of the tibiae were scanned using peripheral quantitative computed tomography (pQCT). The total bone mineral density was measured at 3 mm from the edge of the proximal metaphysis of tibiae. For measurement of shear strength between bone and implants, after removal of soft tissues from femora and fixing them in a central hole (4.5 mm diameter) of a square wooden base with bone cement, the shear strength of bone-implant interface was measured by applying vertical load to the implant, as previously described (1, 2). The shear strength was measured at the peak force when the implant loosened from the bone.

The results were calculated as the mean ± SEM. ANOVA was used to analyze the effect of drugs and HA-coatings. The significance of differences between groups was determined by Fisher’s least significant difference multiple comparison test. Significance was defined as p<0.05.

RESULTS

The total bone mineral density (BMD) in the saline-treated OVX group was significantly lower than that in the control group (p<0.01). However, by administration of alendronate, the total BMD in the OVX group was significantly greater than that in the saline-treated OVX (p=0.001), and achieved the level of that in the control group. Vitamin D treatment was made with little or no improvement in the total BMD. The values of the total BMD in the combined treatment with alendronate and vitamin D were similar to that in alendronate alone treatment (Fig. 1).

The shear strength of HA-coated implants was approximately 40% less in the saline-treated group than in the control group (p=0.042). However, alendronate treatment significantly enhanced the shear strength of HA-coated implants in the OVX group compared with that in the saline-treated OVX group. Vitamin D treatment did not improve the shear strength of both implants in the OVX group. Although the shear strength was highest for combined treatment with alendronate and vitamin D, there were no significant differences compared with that of alendronate treatment group (Fig.2).

DISCUSSION

The present study showed that HA-coatings alone did not obtain enough improvement of implant fixation in our animal model of osteoporosis. However, alendronate treatment enhanced bone mineral density and initial fixation of bone and HA-coated implants in short-term such as 4 weeks after implantation in osteoporotic rats. On the other hand, the effect of vitamin D and the synergistic effect of alendronate and vitamin D on bone mineral density and implant fixation were not shown in osteoporotic condition. We also found that alendronate did not enhance Ti implant stability in osteoporosis. There were some limitations to the present study. First, we did not analyze whether alendronate enhances long-term implant fixation. Second, both implants that we used were smooth surface. If rough surface implant is used, alendronate may enhance implant fixation even in Ti implants.

Our results suggest that the possibility of Alendronate use to increase early HA-coated total joint implant fixation for elderly patients with osteoporosis if these patients do not receive treatment for osteoporosis preoperatively. Future studies are needed to investigate whether alendronate enhances long-term implant fixation and rough surface implant fixation in osteoporosis.

REFERENCES


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