INTRODUCTION

The morbidity of idiopathic osteonecrosis of the femoral head (ION) is increasing every year and over 7,000 patients are treated for ION annually in Japan. Many patients with ION suffer from secondary osteoarthritis of the hip joints due to collapse of the femoral head and have to undergo surgical treatment. Preventing femoral head collapse is essential when treating this bone ischemic disease. Little is known about the mechanism of femoral head collapse after osteonecrosis. Therefore, this study analyzed the trabecular architecture and mineralization in the rat femoral head after traumatic osteonecrosis to reveal the mechanism of the collapse.

MATERIALS AND METHODS

The circulation of the rat femoral head was disrupted using Norman’s procedure [1]. The histological findings of the femoral head showed empty lacunae in the trabeculae on day 7 after the circulatory disruption. Bone regeneration after osteonecrosis, so-called “additional bone formation”, was identified by day 42. The three-dimensional geometry of trabecular bone of the femoral heads was analyzed on days 7, 21, and 42 after circulatory disruption using micro-CT (SMX-130CT-SV, Shimazu, Japan) and a histomorphometry technique that uses our own software. To determine the degree of mineralization of bone (DMB), we developed a phantom containing K$_2$HPO$_4$ solutions of known concentrations Fig. 1. Since there was a linear relationship between the CT number and the concentration of the K$_2$HPO$_4$ solution in the phantom using the least squares method. When we measured the DMB of a K$_2$HPO$_4$ solution of known concentration, the average error of this measuring system was 3.99% and the maximum error was 9.82%. Each 0.95-mm$^3$ cube in the femoral head was subjected to histomorphometry analysis and the DMB was measured. Histomorphometry was performed using the parallel plate model of Parfitt [2] to measure bone volume (BV/TV [%]), trabecular thickness (Tb.Th [µm]), trabecular number (Tb.N [1/mm]), and trabecular space (Tb.Sp [µm]). We defined regions with DMB > 0.8 g/cm$^3$ as bony regions and areas with DMB < 0.8 g/cm$^3$ as marrow regions.

RESULTS

The femoral head with osteonecrosis lost connectivity between trabeculae, which resulted in widening of the trabecular space (Tb.Sp [µm]) (Fig. 3, Table 1). Bone volume (BV/TV [%]) and trabecular thickness (Tb.Th [µm]) decreased transiently after osteonecrosis and increased significantly during additional bone formation after osteonecrosis (Fig. 4). The DMB of the marrow region also showed a transient decrease at day 7 after the circulatory disruption; however, the values at days 21 and 42 indicated a gradual increase during additional bone formation (Table 1).

DISCUSSION

We successfully demonstrated the three-dimensional changes that occur in trabecular architecture and mineralization after osteonecrosis in the rat femoral head using micro-CT and a novel mineralization measuring system using a phantom made of K$_2$HPO$_4$ solution. Our results show a transient decrease in trabecular bone volume and mineralization just after bone ischemia, before vascular regeneration, and a subsequent increase over time during additional bone formation. These changes in bone volume and mineralization might induce disproportional changes in the hardness and elasticity of trabecular bone, resulting in subsequent femoral head collapse. Mechanical tests of the three-dimensional trabecular architecture after osteonecrosis are now in progress. These data will be useful for revealing the mechanism of femoral head collapse, which is a target in ION treatment.

REFERENCES