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LETTER TO THE EDITOR

Comment on: Osteogenic abilities of bone marrow stromal cells are not defective in patients with osteonecrosis

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Dear Editor,

We have recently read the article entitled "Osteogenic abilities of bone marrow stromal cells are not defective in patients with osteonecrosis" by Yoo et al. [4]. The purpose of the research was to investigate whether the proliferation activities and osteogenic capacities of bone marrow stromal cells (BMSCs) are depressed in patients with osteonecrosis of the femoral head (ONFH). They concluded that the osteogenic potentials of BMSCs are not defective in patients with ONFH.

Although detailed pathogenesis and aetiology are still unknown, several risk factors concerning ONFH have been elucidated, including alcohol abuse, corticosteroid treatment and thrombophilia-hypofibrinolysis disorders. Previous studies on the mechanism of ONFH are mostly focussed on extra-cellular factors; however, cellular apoptosis and behavioural alterations of stem and progenitor cells have attracted great attention recently. Hernigou et al. found a decrease in the mesenchymal stem-cell pool in the proximal femur of patients with corticosteroid-induced osteonecrosis and suggested that glucocorticosteroids had an adverse effect on bone by decreasing the progenitors [2]. Similarly, Gangji et al. found abnormalities in the replicative capacity of osteoblastic cells in the proximal femur of patients with osteonecrosis of the femoral head [1]. Although we could not currently consider ONFH as a stem-cell or progenitor-related disease, the pathogenesis should consider the possibility of a very close relationship with the cellular origin.

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Yoo et al. found that osteogenic abilities of BMSCs are not defective in patients with ONFH, conflicts with previous reports, and consequently makes us more confused about the complex mechanism. We found that the BMSCs that Yoo et al. used were from the iliac crests of patients with ONFH, while the donor sites of BMSCs from previous reports were from proximal femur or near the site of osteonecrosis [1–3]. We think the difference in the donor sites of BMSCs might contribute to the discrepancy, although to our knowledge there is no direct evidence to support our hypothesis. In principle, the micro-environment of the iliac crests where BMSCs exist could not reflect the true condition in the necrotic zone, and probably BMSCs from the necrotic site would be more representative.

Cellular and extra-cellular events might combine to cause the onset of ONFH, and it is a little arbitrary to draw these conclusions from the method of Yoo et al. Further exploration should is needed to clarify the biological behaviour of BMSCs in the necrotic site and remote areas.

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