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Bone fragility in diabetes mellitus and regulation of glucose metabolism by bone

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Accumulating evidence has shown that the risk of osteoporotic fractures is increased in patients with diabetes mellitus although they have normal or increased bone mineral density (BMD). Since the incidence of osteoporotic fracture is related with morbidity and mortality, it became social issue especially in industrial countries. Advanced glycation end products (AGEs) are shown to be involved in diabetes mellitus-related bone fragility. AGEs inhibits osteoblastic differentiation. Moreover, AGEs-collagen crosslinks in bone matrix weaken bone strength. Recently, it was found that cortical porosity exists in diabetic patients with history of osteoporotic fractures. In clinical settings, it is required to clarify how to detect the diabetes-related bone fragility because BMD measurement is not useful for screening. On the other hand, it is reported that bone regulates glucose metabolism vice versa. Osteocalcin produced by osteoblasts stimulates insulin secretion in pancreatic beta-cells as well as adiponectin in adipocytes, resulting in preventing impaired glucose tolerance and fat accumulation. Most recently, osteocalcin is reported to stimulate secretion of glucose-like peptide-1 in small intestine. The author will review the diabetes-related bone fragility as well as the endocrine function of osteocalcin.

Biography

Ippei Kanazawa obtained PhD degree in 2009 from Shimane University Graduate School of Medicine and completed Postdoctoral studies at McGill University in Canada. He is now an Assistant Professor of the division of Endocrinology and Metabolism in Shimane University Faculty of Medicine. He has published more than 45 papers in international journals and serving as an Editorial Board Member of *Journal of Diabetes & Metabolism.*

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