ABSTRACT

Hyperglycemia caused reduction of cortical bone thickness in streptozotocin-induced diabetic rat

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Introduction: Diabetes is a chronic metabolic disease characterized by hyperglycemia due to insulin deficiency (type 1 diabetes) or insulin insensitivity/resistance (type 2 diabetes). The significant metabolic changes that occur in diabetes also affect the skeleton and cause bone loss and/or altered bone matrix and strength, thereby increasing the risk of fracture. Hyperglycemia can alter cellular metabolic processes through the formation of advanced glycation end products (AGEs), which caused disregulation of cytokines such as TNF-α, M-CSF and RANKL, which is thought to be the underlying mechanism in the decrease of bone density. This study aimed to see the bone loss caused by hyperglycemia in rats, using cortical bone thickness as a parameter.

Methods: This study was an analytical experimental study, which was conducted in a laboratory. The sample of this study was rat (Rattus norvegicus). The sample was obtained from Laboratory Animal Unit of Department of Biochemistry in Airlangga University. Diabetes in the rats were induced using streptozotocin. The diabetic rats were then sacrificed 11 days later, and the left femur bone was obtained. The bone was the decalcified for 1 week, and then sent to Histology Laboratory of Airlangga University to be made into histological slides. Bone cortical thickness was measured at 10 different points and then averaged.

Results: The mean of cortical bone thickness were 32.43 ± 2.65 µm in non diabetic rats, and 26.64 ± 2.89 µm in streptozotocin-induced diabetic rats. The diabetic rats had lower mean of cortical bone thickness than non diabetic rats by 5.79 µm.

Conclusion: Cortical bone thickness in streptozotocin-induced diabetic rats are lower compared to non diabetic rats.

Keywords: hyperglycemia, diabetes, bone, cortical thickness, streptozotocin, rat