Diabetes decreases hippocalcin expression in focal cerebral ischemia

P.O. Koh, D.J. Park
Department of Veterinary Medicine, College of Veterinary Medicine, Institute of Agriculture and Life Science, Gyeongsang National University, South Korea

Stroke is a major cause of disability and death in adults. Hyperglycemia causes intracellular calcium imbalance after ischemic insult, aggravates cytochrome c release into cytosol and activates caspase-3, and ultimately triggers apoptosis. Hippocalcin is a neuronal calcium-sensor protein that acts as a calcium buffer to regulate the intracellular concentration of Ca^{2+}. This study was investigated to elucidate hippocalcin protein expression of the cerebral cortex during ischemic brain injury between non-diabetic and diabetic animals. Adult male rats were injected with streptozotocin (40 mg/kg) via the intraperitoneal route to induce diabetes and underwent surgical middle cerebral artery occlusion (MCAO) 4 weeks after streptozotocin treatment. Cerebral cortex tissues were collected 24 h after MCAO. A proteomic approach and Western blot analysis revealed that hippocalcin protein was significantly decreased in diabetic animals with MCAO injury compared to diabetic-only and MCAO-only animals. The decrease of hippocalcin in hyperglycemic condition suggest that hyperglycemia leads to intracellular calcium imbalance by regulating hippocalcin expression levels in ischemic brain injury.