Circulating lncRNAs HIF1A AS2 and LINLK A: Role and Relation to Hypoxia Inducible Factor 1Ŭ in Cerebral Stroke Patients

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Abstract

Long noncoding RNAs (lncRNAs) have been recently recognized as key players of gene expression in cerebral pathogenesis. Thus, their potential use in stroke diagnosis, prognosis, and therapy is actively pursued. Due to the complexity of the disease, identifying stroke-specific lncRNAs remains a challenge. This study investigated the expression of lncRNAs HIF1A-AS2 and LINK-A, and their target gene hypoxia-inducible factor-1 (HIF-1) in Egyptian stroke patients. It also aimed to determine the molecular mechanism implicated in the disease. A total of 75 stroke patients were divided into three clinical subgroups, besides 25 healthy controls of age-matched and sex-matched. Remarkable upregulation of lncRNA HIF1A-AS2 and HIF1-Ŭ along with a downregulation of lncRNA LINK-A was noticed in all stroke groups relative to controls. Serum levels of phosphatidylinositol 3-kinase (PI3K), phosphorylated-Akt (p-Akt), vascular endothelial growth factor (VEGF), and angiopoietin-1 (ANG1) as well as their receptors, malondialdehyde (MDA), and total antioxidant capacity (TAC) were significantly increased, whereas brain-derived neurotrophic factor (BDNF) levels were significantly decreased particularly in hemorrhagic stroke versus ischemic groups. Eventually, these findings support the role of lncRNAs HIF1A-AS2 and LINK-A as well as HIF1-Ŭ in activation of angiogenesis, neovascularization, and better prognosis of stroke, especially the hemorrhagic type.

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