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POSTER

ETV-2 activated proliferation of endothelial cells and attenuated acute hindlimb ischemia in mice

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Abstract

Ischemia is the reduction of blood flow to tissues by injury of blood vessels. Depending on the sites of tissues and grade of ischemia, ischemia can cause many serious complications. This study aimed to evaluate the effects of the E-twenty six (ETS) factor Ets variant 2 (ETV2) gene expression in angiogenesis and the effect of ETV2 gene therapy in a mouse model of hindlimb ischemia. The role of ETV2 on endothelial cell proliferation was evaluated in vitro. Knockdown of ETV2 expression was done using short hairpin RNA (shRNA) lentiviral viral particles. The ETV2 viral vector was injected into the skeletal muscles at the ligated and burned sites of the hindlimb and evaluated for its efficacy as a gene therapy modality for ischemia. Vascular regeneration in mice was indirectly evaluated by changes in mouse survival, necrotic grades of the leg, normal blood oxygen saturation level (SpO_2), and blood flow by trypan blue injection assay. Preliminary data showed that ETV2 expression played a role in angiogenesis of endothelial cells. ETV2 overexpression could trigger and stimulate proliferation of skeletal endothelial cells. In vivo knockdown of ETV2 expression inhibited the auto-recovery of ischemic hindlimb, while overexpression of ETV2 helped to rescue leg loss and reduce necrosis, significantly improving angiogenesis in hindlimb ischemia. Our findings demonstrate that ETV2 gene therapy is a potentially effective modality for vascular regeneration.

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