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Syndecan -1 as Regulator of Ischemic-Reperfusion Damage Limitation in Experiment

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Abstract : Brain neuroplasticity is associated with blood-brain barrier vascular endothelial proteoglycans and post-stroke microglial activation. The study of the mechanisms of reperfusion injury limitation by remote ischemic postconditioning (RC) is of interest due to the effects on functional recovery after cerebral ischemia. The goal of the study is the assessment of the role of syndecan-1 (SDC-1) in restriction of ischemic-reperfusion injury on middle cerebral artery model in rats using RC protocol. Randomized controlled trials were conducted. Ischemia was performed by middle cerebral artery occlusion by Belayev L. (1996) on the Wistar rat-males (n= 87) weighting 250 ± 50 g. under general anesthesia (Zoletil 100 и Xylazine 2%). Syndecan-1 (SDC-1) concentration difference in plasma samples of false operated animals and animals with brain ischemia was 30% (30 min. MCAo: 41.4 * ± 1.3 ng/ml). SDC-1 concentration in animal plasma samples with ischemia + RC protocol was 112% (30 min MCAo+ RC): 67.8**± 5.8 ng/ml). Calculation of infarction volume in the ischemia group revealed brain injury in 31.97 ± 2.5%; the volume of infarction was 13.6 ± 1.3% in 30 min. MCAo + RC group. Swelling of tissue in the group 30 min. MCAo + RC was 16 ± 2.1%; it was 47 ± 3.3%. in 30 min. MCAo group. Correlation analysis showed a high direct correlation relationship between infarct area and muscle strength in the right forelimb (KK=0.72) in the 30 min. MCAo + RC group. Correlation analysis showed very high inverse correlation between infarct area and capillary blood flow in the 30 min. MCAo + RC group ($p < 0.01$; $r = -0.98$). We believe the SDC-1 molecule in blood plasma may play role of potential messenger of ischemic-reperfusion injury restriction mechanisms. This leads to infarct-limiting effect of remote ischemic postconditioning and early functioning recovery.

Keywords : ischemia, MCAo, remote ischemic postconditioning, syndecan-1
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