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Human umbilical cord mesenchymal stem cells reduce H9C2 cells apoptosis via PI3K-akt pathway by paracrine activity

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Introduction & Objective: As limitations of Mesenchymal Stem Cells (MSCs) transplantation, paracrine activity of MSCs is brought into focus. The study aimed to explore the effect of human Umbilical Cord MSCs (UC-MSCs) paracrine in H9C2 cells apoptosis and its related mechanisms.

Method: After Conditioned Medium (CM) of UC-MSCs preparation, antibody arrays were used to determine the difference of cytokines between normoxia and hypoxia CM of UC-MSCs. After apoptosis was induced by Hypoxia and Serum Deprivation (H/SD), H9C2 cells were treated with normoxia and hypoxia CM, normoxia and hypoxia exosomes, LY294002 and rapamycin respectively. Cell viability was detected by the CCK-8 assay. Apoptosis was detected by Annexin V-FITC/PI and Hochest staining. Western blot was used to detect expression level of caspase-3, LC3B, P62, Beclin-1, Akt, p-Akt, mTOR and p-mTOR. Immunofluorescence analysis was used to detect LC3B expression.

Result: Hypoxic preconditioning enhanced UC-MSCs viability changed the composition of UC-MSCs secretome and increased the secretion of exosomes. Both CM and exosomes of UC-MSCs could reduce H9C2 cells apoptosis, increase H9C2 cells viability, up-regulated expression of P62 and down-regulated expression of cleaved caspase-3, LC3B II/I and Beclin-1. Hypoxic preconditioning could strengthen these effects. Hypoxia conditioned mediums and exosomes increased level of P-Akt/Akt and P-mTOR/mTOR. The anti-apoptotic effect of conditioned mediums and exosomes could be attenuated by treatment with LY294002 and rapamycin.

Conclusion: UC-MSCs could reduce H9C2 cells apoptosis induced by H/SD through regulating autophagy via PI3K-Akt pathway by paracrine activity. Hypoxia preconditioning strengthened this anti-apoptosis effect through enhancing paracrine activity of UC-MSCs.

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