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The Role of Autophagy Modulation in Angiotensin-II Induced Hypertrophy

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Abstract : Autophagy plays an important role in cardiac hypertrophy, which is one of the most common causes of heart failure in the world. This self-degradative catabolic process, responsible for protein quality control, balancing sources of energy at critical times, and elimination of damaged organelles. The autophagic activity can be triggered by starvation, oxidative stress, or pharmacological agents, like rapamycin. This induced autophagy can promote cell survival during starvation or pathological stress. In this study, it is investigated the effect of the induced autophagic process on angiotensin induced hypertrophic H9c2 cells. In our study, it is used H9c2 cells as an in vitro model. To induce hypertrophy, cells were treated with 10000 nM angiotensin-II, and to activate autophagy, 100 nM rapamycin treatment was used. The following groups were formed: 1: control, 2: 10000 nM AT-II, 3: 100 nM rapamycin, 4: 100 nM rapamycin pretreatment then 10000 nM AT-II. The cell viability was examined via MTT (cell proliferation assay) assay. The cells were stained with rhodamine-conjugated phalloidin and DAPI to visualize F-actin filaments and cell nuclei then the cell size alteration was examined in a fluorescence microscope. Furthermore, the expression levels of autophagic and apoptotic proteins such as Beclin-1, p62, LC3B-II, Cleaved Caspase-3 were evaluated by Western blot. MTT assay result suggests that the used pharmaceutical agents in the tested concentrations did not have a toxic effect; however, at group 3, a slight decrement was detected in cell viability. In response to AT-II treatment, a significant increase was detected in the cell size; cells became hypertrophic. However, rapamycin pretreatment slightly reduced the cell size compared to group 2. Western blot results showed that AT-II treatment-induced autophagy, because the increased expression of Beclin-1, p62, LC3B-II were observed. However, due to the incomplete autophagy, the apoptotic Cleaved Caspase-3 expression also increased. Rapamycin pretreatment up-regulated Beclin-1 and LC3B-II, down-regulated p62 and Cleaved Caspase-3, indicating that rapamycin-induced autophagy can restore the normal autophagic flux. Taken together, our results suggest that rapamycin activated autophagy reduces angiotensin-II induced hypertrophy.

Keywords : angiotensin-II, autophagy, H9c2 cell line, hypertrophy, rapamycin
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