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Exogenous HGF prevents cardiomyocytes from apoptosis after hypoxia via up-regulating cell autophagy

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Hepatocyte growth factor (HGF) is widely known as a protective factor in ischemic myocardium, however the mechanism remains unclear. Autophagy at early stage of hypoxia has been demonstrated to play an important role in protecting myocardium both *in vivo* and *in vitro*. We performed this study to investigate the association between the protective effect of HGF and autophagy. We found that autophagy in neonatal rat ventricular myocytes (NRVMs) increased at early stage after hypoxia and HGF release was consistent with the change of autophagy. Then we added exogenous HGF to the cells and enhanced autophagy was detected, while neutralizing HGF got opposite effects. However, after inhibition of autophagy using 3-methyladenine, apoptosis of myocytes increased indicating the protective effect of HGF was autophagy-dependent. This may be associated with clearance of injured mitochondria as the marker of mitochondrial autophagy, parkin, was induced when HGF was added to cell medium. Our results provided insight into a potential mechanism in which exogenous HGF prevented NRVMs from apoptosis after hypoxia. Upregulation of parkin through administration of exogenous HGF may be a potential therapeutic strategy for myocytes ischemia.

Biography

Yunle Wang is a PhD student in Nanjing Medical University. She is doing her PhD in Cardiology and works for the team of Prof. Zhijian Yang. Her studies focus on the protection effect of hepatocyte growth factor on myocardial infarction and the function of autophagy in myocardial ischemia disease.

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