The Effect of Contrast Media on Renal Cell Autophagy

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Backgrounds: Contrast media is a substance to enhance the contrast of structures or fluids within the body in medical imaging, and it is used to assist the improvement of X-ray image and MR signal. The contrast medium injected into body is excreted via kidney. Therefore, contrast-induced nephropathy (CIN) is the most common cause of acute kidney injury in hospitalized patients. The mechanisms of CIN are known as vasoconstriction and reduction in renal blood flow, following the increasing of ROS production and renal cell apoptosis. Autophagy is the mechanism by which cells consume parts of themselves to survive starvation and stress. Recently, many studies have reported that both apoptosis and autophagy can be induced under some specific stresses. However, the role of autophagy on contrast-induced cell damage has not been studied. Materials and Methods: Telebrix 35 is a hyper- and visipaque is a iso-osmolar contrast media respectively. In our studies, pig kidney epithelial (PK1) cells were treated theses two different contrast media and the LC3-II formation from LC3-I were detected by western blotting and immunofluorescence stain. Results: Both contrast media, telebrix 35 and visipaque, could induce autophagy on PK1 cells. Both contrast media increased LC3-II formation and the mRNA level of Atg-6 and Atg 5 assessed by real-time PCR. Interestingly, visipaque enhanced more LC3-II and autophagosomes formation than telebrix 35 on PK1 cells. The more autophagy induced by visipaque could significantly protect PK1 cells from contrast media-induced apoptosis when compare to Telebrix 35. Moreover, we found that the increase of autophagy by these contrast media might be through the ROS and mTOR pathways. Conclusion: Contrast media can induce autophagy on PK1 cells and this enhanced autophagy may protect PK1 cells from apoptosis. These results will provide clearer mechanisms and efficient methods for prevention renal cells from the contrast-induced nephropathy.