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The Effect of Hepatic Ischemia and Reperfusion on Energy Metabolism in Rats

Jeong Cheol°, Tai-Soon Cho and Sun-Mee Lee

Sung Kyun Kwan University

It was reported that ATP depletion occurs and accelerates cell damage during ischemia and reperfusion. To determine the mechanism of cell damage, the change of energy metabolim in liver was studied during ischemia/reperfusion. The groups were divided into four categories : sham-operated group, ischemia/reperfusion group, and two types of ATP-$MgCl_2$ treatment groups(one was treated during ischemia and the another during reperfusion). Rats were administered intravenously saline or ATP-$MgCl_2$. Rats were anesthetized and blood vessels to the left and median lobes of the liver were occluded. After 60min of ischemia, the clamp at those vessels were removed. After ischemia, one and five hours after reflow, energy metabolites(ATP, ADP, AMP, inosine, adenosine, hypoxanthine, xanthine) in liver were measured with HPLC. To observe mitochondrial function, aterial keton body ratio in blood and mitochondrial glutamate dehydrogenase activity in liver were measured. And lipid peroxidation was measured to evalutate the involvement of free radicals. In this study, ATP and ADP were catabolized to their metabolites(AMP, inosine, adenosine, hypoxanthine, xanthine) during ischemia and they resynthesized ATP and ADP during reperfusion. But total purine base were not restored to level of normal rat. The main source of resynthesizing ATP and ADP was AMP. In both ATP-$MgCl_2$ treated groups, mitochondrial function was protected and lipid peroxidation was significantly reduced. Our findings suggest that ischemia/reperfusion impairs hepatic energy metabolism.