Effects of acute administration of acetyl-L-carnitine on hemodynamic function and infarct size in ischemic-reperfused isolated heart

M. Najafi1,*, S. Ghaffary Eyrdmousa2

1Department of Pharmacology, School of Pharmacy, Tabriz University of Medical Sciences, Tabriz, I.R. Iran
2School of Pharmacy, Tabriz University of Medical Sciences, Tabriz, I.R. Iran

Background and Aims: Acetyl-L-carnitine (ALC) plays a fundamental role in the production and dissemination of cellular energy as well as in the regulation of metabolic pathways because it is a co-factor required for transport of long-chain fatty acids through the mitochondrial membrane in cardiomyocytes. In this study, effects of acute administration of ALC on hemodynamic functions and infarct size were investigated in isolated rat heart.

Methods: The isolated hearts were perfused by a modified Krebs-Henseleit solution during 30 min regional ischemia followed by 120 min reperfusion (control) or enriched Krebs solution with 0.375, 1.5 and 3 mM of ALC. Hemodynamic parameters including LVDP, HR, rate pressure product (RPP) and coronary flow rate (CFR) were recorded. The infarct size was determined by using a computerized planimetry package.

Results: During both ischemia and reperfusion phases, acute perfusion of ALC increased HR and RPP in comparison with the control group. The effect was significant by 3 mM of ALC (P<0.001), while LVDP and CFR did not show any significant changes. In the control group, the infarct size was 25±3%, however, ALC (0.375, 1.5 and 3 mM) reduced it to 13±2, 15±3 and 14±4%, respectively (P<0.05).

Conclusions: ALC produced protective effects against cardiac ischemia/reperfusion injuries as improvement of some determinants of posts ischemic hemodynamic functions and reduction of infarct size. Among the potential cardioprotective mechanisms for ALC, increasing in glucose oxidation then reducing lactate production, reduction of toxic fatty acid metabolites and removing free radicals from the myocytes are more relevant.

Keywords: Acetyl-L-carnitine; Hemodynamic function; Ischemia; Reperfusion; Rat