Genetics and Molecular Research

Impact of citrate pretreatment on ventricular arrhythmia and myocardial capase-3 expression in ischemia/reperfusion injury

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Genet. Mol. Res. 15 (4): gmr15048848
Received June 2, 2016
Accepted August 10, 2016
Published October 17, 2016
DOI http://dx.doi.org/10.4238/gmr15048848

ABSTRACT. Ischemia/reperfusion (I/R) injury often triggers ventricular arrhythmia. Citrate binds calcium ions, forming a soluble calcium citrate complex that may reduce I/R injury by affecting calcium ion concentration. We tested the effects of citrate pretreatment on ventricular heart rate and related factors in a rat I/R model. Fifty male Sprague Dawley rats weighing 350-400 g were randomly divided into equally sized control (A), model (B), and 0.1 M (C), 0.05 M (D), and 0.025 M (E) citrate groups. An I/R model was established by ligating the left anterior descending coronary artery. Serum calcium ion concentration was measured before and after citrate treatment.

Triphenyltetrazolium chloride staining and spectrophotometry were used to determine infarction area and caspase-3 protein levels in myocardial tissue, respectively. Polymerase chain reaction was performed to test myocardial calmodulin (CAM) expression. The frequency of ventricular arrhythmia in group B was significantly higher than in the sham surgery group ($P < 0.05$). Citrate pretreatment resulted in lower and higher frequencies than those observed in the model and control groups, respectively, in a dose-independent manner. The most obvious reduction in ventricular arrhythmia was seen in Group D. Serum calcium ion concentration decreased markedly after citrate treatment ($P < 0.05$), with a specific pattern emerging over time. Infarction area and caspase-3 and CAM levels were significantly lower in the citrate groups compared with the model group ($P < 0.05$). Citrate can reduce myocardial cell apoptosis, alleviating ventricular arrhythmia and protecting the myocardium by reducing serum calcium ion concentration and downregulating caspase-3 and CAM expression.

Key words: Citrate; Ventricular arrhythmia; Caspase-3