Magnolol reduces myocardial ischemia/reperfusion injury via neutrophil inhibition in rats.

許準榕

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Abstract

The accumulation of oxygen-free radicals and activation of neutrophils are strongly implicated as important pathophysiological mechanisms mediating myocardial ischemia/reperfusion injury. It has been proven that various antioxidants have cardioprotective effects. Magnolol, an active component extracted from the Chinese medicinal herb Magnolia officinalis, possesses potent antioxidant and free radical scavenging activities. In this study, the cardioprotective activity of magnolol was evaluated in an open-chest anesthetized rat model of myocardial ischemia/reperfusion injury. The results demonstrated that pretreatment with magnolol (0.2 and 0.5 microg/kg, i.v. bolus) at 10 min before 45 min of left coronary artery occlusion, significantly suppressed the incidence of ventricular fibrillation and mortality when compared with the control group. Magnolol (0.2 and 0.5 microg/kg) also significantly reduced the total duration of ventricular tachycardia and ventricular fibrillation. After 1 h of reperfusion, pretreatment with magnolol (0.2 and 0.5 microg/kg) caused a significant reduction in infarct size. In addition, magnolol (0.2 microg/kg) significantly reduced superoxide anion production and myeloperoxidase activity, an index of neutrophil infiltration in the ischemic myocardium. In addition, pretreatment with magnolol (0.2 and 0.5 microg/kg) suppressed ventricular arrhythmias elicited by reperfusion following 5 min of ischemia. In vitro studies of magnolol (5, 20 and 50 microM) significantly suppressed N-formylmethionyl-leucyl-phenylalanine (fMLP; 25 nM)-activated human neutrophil migration in a concentration-dependent manner. It is concluded that magnolol suppresses ischemia- and reperfusion-induced ventricular arrhythmias and reduces the size of the infarct resulting from ischemia/reperfusion injury. This pronounced cardioprotective activity of magnolol may be mediated by its antioxidant activity and by its capacity for neutrophil inhibition in myocardial ischemia/reperfusion.