Microparticle release in remote ischemic conditioning mechanism

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Résumé en anglais	Remote ischemic conditioning (RCond) induced by short periods of ischemia and reperfusion of an organ or tissue before myocardial reperfusion is an attractive strategy of cardioprotection in the context of acute myocardial infarction. Nonetheless, its mechanism remains unknown. A humoral factor appears to be involved, although its identity is currently unknown. We hypothesized that the circulating microparticles (MPs) are the link between the remote tissue and the heart. MPs from rats and healthy humans undergoing RCond were characterized. In rats, RCond was induced by 10 min of limb ischemia. In humans, RCond was induced by three cycles of 5-min inflation and 5-min deflation of a blood-pressure cuff. In the second part of the study, rats underwent 40 min myocardial ischemia followed by 2 h reperfusion. Infarct size was measured and compared among three groups of rats: 1) myocardial infarction alone (MI) (n = 6); 2) MI + RCond started 20 min after coronary ligation (n = 6); and 3) MI + injection of RCond-derived rat MPs (MI + MPs) (n = 5). MPs from endothelial cells (CD54(+) and CD146(+) for rats and humans, respectively) and procoagulant MPs (Annexin V(+)) markedly increased after RCond, both in rats and humans. RCond reduced infarct size (24.4 ± 5.9% in MI + RCond vs. 54.6 ± 4.7% in MI alone; $P < 0.01$). Infarct size did not decrease in MI + MPs compared with MI alone (50.2 ± 6.4% vs. 54.6 ± 4.7%, not significantly different). RCond increased endothelium-derived and procoagulant MPs in both rats and humans. However, MP release did not appear to be a biological vector of RCond in our model.
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