Effect of ischemic preconditioning on regional release of inflammation markers

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摘要

Abstract

Systemic markers of inflammation may be increased in patients after percutaneous coronary intervention. In the present study, we evaluated whether IP (ischaemic preconditioning) attenuated inflammation by activating KATP (ATP-sensitive potassium) channels in patients undergoing coronary angioplasty. Patients (n=36) undergoing angioplasty of a major left coronary artery were allocated randomly to one of four groups: a control group, a group receiving nicorandil (an agonist of KATP channels), an IP group or an IP group pretreated with glibenclamide (an antagonist of KATP channels). To measure the release of sCD40L, P-selectin and myeloperoxidase from the ischaemic region, blood samples were drawn simultaneously from the ascending aorta and the great cardiac vein before and 15 min after coronary angioplasty. At 15 min after angioplasty, a significant increase in sCD40L and P-selectin levels in the great cardiac vein in the control group was observed. IP- and nicorandil-treated patients did not show a significant change in sCD40L and P-selectin levels in response to angioplasty. However, the IP-induced attenuation of sCD40L and P-selectin release was abolished by administering glibenclamide. The change in myeloperoxidase levels mirrored those of sCD40L and P-selectin. The levels of inflammatory markers in the aorta remained stable throughout the study. Patients undergoing angioplasty had increased sCD40L and P-selectin levels in the ischaemic region. In conclusion, IP abolished angioplasty-induced myeloperoxidase release by preventing activated platelet-induced P-selectin release via a KATP-channel-initiated pathway. Therefore, in addition to its primary effect on cardioprotection, IP may also provide beneficial anti-inflammatory effects on the interaction between platelets and neutrophils