Cardioprotective effect of atorvastatin alone or in combination with remote ischemic preconditioning on the biochemical changes induced by ischemic/reperfusion injury in a mutual prospective study with a clinical and experimental animal arm

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Abstract:

Background: Atorvastatin and remote ischemic preconditioning (RIPC) have beneficial cardiovascular protective effects. The aim of the study was to investigate possible effect of this drug alone and in combination with RIPC on the biochemical changes induced by ischemic/reperfusion injury (I/R) in a combined study with a clinical and experimental animal arm. Methods: Thirty consecutive patients undergoing elective percutaneous coronary intervention (PCI) were divided into three groups (10 each): group I (control group without any preconditioning), group II (patients who were maintained on atorvastatin (80 mg/day) for one month before PCI), and group III (similar to group II but PCI was preceded by RIPC). On the other hand, sixty adult male New Zealand white rabbits were divided into 6 groups (10 each): group I (control), group II (sham), group III (I/R as 30 min ischemia followed by 120 min reperfusion), group IV (regular atorvastatin 10 mg/kg for 40 days orally followed by I/R), group V (I/R preceded by RIPC) and group VI (similar to group IV but I/R was preceded by RIPC). Tumor necrosis factor-α (TNF-α), interleukin-6 (IL-6), nitric oxide (NO), troponin I (cTnI), creatine kinase MB (CK-MB) and C-reactive protein (CRP) were measured in blood for all study groups. Results: Clinical and experimental parts showed that groups with RIPC combined with atorvastatin pre-treatment showed a synergistic protective effect against I/R injury as evidenced by significant reduction (P < 0.001) in the levels of TNF-α, cTnI (in patients) and IL-6, CK-MB and CRP (in rabbits) while the level of NO was significantly (P < 0.001) increased compared with other groups. Conclusions: Pretreatment with atorvastatin combined with RIPC can exert a synergistic cardioprotective effects by reducing the possible biochemical changes related to ischemic reperfusion injury.

Published In:

International Journal of Cardiology, vol. 222, pp. 866–873