

in 2 (10.0%) patients in the loading dose group, 2 (10.0%) patients in the regular dose group, 3 (15.0%) patients in the control group, respectively ($p=0.855$).

Conclusions Atorvastatin loading in patients with STEMI undergoing primary PCI may not have protective effects on coronary endothelial function, inflammation, and MACE.

Related pharmaceutical clinical research

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EFFECTS OF STATIN LOADING BEFORE PRIMARY PCI ON CORONARY ENDOTHELIAL FUNCTION AND INFLAMMATION

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Objectives The Novel approaches for preventing or limiting events (NAPLIES) and The Atorvastatin for Reduction of Myocardial Damage during Angioplasty (ARMYDA) studies demonstrated a beneficial effect of statin loading in preventing major adverse cardiac events (MACE) after elective percutaneous coronary intervention (PCI) for stable angina, unstable angina, non-ST-segment-elevation myocardial infarction (NSTEMI). The so called 'pleiotropic effects' of statins include modulation of endothelial function, inhibition of inflammation, and attenuation of thrombosis, all of which could provide clinical benefits in the setting of elective PCI by reducing postprocedural incidence of myocardial and MACE. So far, the efficacy of atorvastatin loading in patients with acute ST-segment-elevation myocardial infarction (STEMI) undergoing primary PCI has not been confirmed. Also, whether the 'pleiotropic effects' of statins could explain the possible mechanism (s) needs to be discussed. This study sought to explore potential protective effects of statin loading before primary PCI on coronary endothelial function, inflammation, and MACE.

Methods A total 60 patients with STEMI were randomised into loading dose group (80 mg atorvastatin before PCI, $n=20$), regular dose group (20 mg atorvastatin before PCI, $n=20$), and control group (without atorvastatin before PCI, $n=20$). All patients received primary PCI and routine treatment. The plasma samples were collected before, immediately after, 6 h after and 24 h after PCI in all the patients. Plasma concentrations of endothelial nitric oxide synthase (eNOS), Nitric Oxide (NO), interleukin-6 (IL-6), tumour necrosis factor (TNF- α), intercellular adhesion molecule-1 (ICAM-1) were tested by ELISA. The results of coronarography, electrocardiogram, myocardial enzyme, high-sensitivity C-reactive protein (hs-CRP), amino terminal-pro brain natriuretic peptide (NT-proBNP), echocardiography, MACE, and the safety of statin loading were also collected.

Results Plasma eNOS immediately and 24 h after PCI were higher in the regular dose group ($p<0.05$). Plasma eNOS before and 24 h after PCI, along with plasma NO at any time point did not show significant differences among the 3 groups. Plasma IL-6 before PCI were lower in the loading dose group (90.773 ± 7.646 pg/ml vs 95.592 ± 4.269 pg/ml vs 94.324 ± 3.692 pg/ml, $p=0.023$). Plasma IL-6 after PCI, plasma TNF- α and ICAM-1 at any time point did not show significant differences among the 3 groups. MACE occurred