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EFFECT OF ATORVASTATIN (LIPITOR) ON MYOCARDIAL APOPTOSIS AND CASPASE-8 ACTIVATION FOLLOWING CORONARY MICROEMBOLISATION

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We determined the effect of atorvastatin on myocardial apoptosis and caspase-8 activation following coronary microembolisation (CME) in a rat model. For this, 50 rats were randomly and equally divided into CME; sham-operated (control); atorvastatin lavage; gastric-lavage control; and caspase-8 inhibitor (CHO) groups. In CME animals, a microembolisation ball was injected through the left ventricle. Sham animals were injected with normal saline (NS). Atorvastatin group received atorvastatin gastric lavage once-a-day, 1-week before surgery. Gastric-lavage controls had similar lavage with NS. CHO group was intraperitoneally-injected (CHO-10 mg/kg) 30 min before surgery. Cardiac indices in each group were determined by echocardiography 6 h postoperatively. TUNEL assay and western blot were used for myocardial apoptosis and expression of caspases-3/-8, respectively. Echocardiography data show that left ventricular ejection fraction (LVEF) in CME group was significantly decreased ($p<0.05$) compared with sham controls. Besides, left ventricular shortening fraction (FS) and cardiac output (CO) were also decreased with an increase in left ventricular end-diastolic dimension (LVEDd). Atorvastatin and CHO animals had significantly improved ($p<0.05$) cardiac function compared with CME group. Myocardial apoptosis and activation levels of caspases-3/-8 were significantly increased ($p<0.05$) compared with sham; myocardial apoptosis and activation levels of caspases-3/-8 were significantly decreased ($p<0.05$) in atorvastatin and CHO groups compared with CME group. In conclusion, atorvastatin pretreatment suppressed post-CME myocardial apoptosis and improved cardiac function through the blockade of a myocardial death receptor-mediated apoptotic pathway.