model was induced by ligation of left coronary artery combined with cold stress of 4°C,8 h/day for four consecutive days. Rats in A group were gavaged with atorvastatin 20 mg/kg/d for 3 days before co-stress was induced and thereafter for four days. Excluding the dead animals of anaesthetic accident, arrhythmia, acute heart failure after surgery, 12 rats were in each group eventually. Cardiac function was assessed by echocardiography; myocardial infarct size was determined by TTC staining; p- PI3K, p-GSK3β, Bim and caspase-3 expression in myocardium was determined by western blotting.

Results It was demonstrated that co-exposure to myocardial ischemia and cold stress could significantly deteriorate the cardiac function and increase the infarct size ($p<0.01$), and this was attenuated by atorvastatin with increased expression of p-PI3K, p-GSK3β, and Bim, caspase3 downregulating ($p<0.01$).

Conclusion It is possible that atorvastatin attenuates cardiac injury induced by co-stress of myocardial ischemia and cold stress through activating PI3K/Akt/GSK3β pathway and decreasing expression of Bim.

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EFFECT OF ATORVASTATIN ON MYOCARDIAL INJURY IN RATS FOLLOWING CO-STRESS OF MYOCARDIAL ISCHEMIA AND COLD

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Objective To investigate the role of atorvastatin in myocardium of rats following co-stress of myocardial ischemia and cold stress, and the relative mechanism.

Methods Seventy male Sprague–Dawley rats were randomly divided to five groups: sham+normal temperature (S group); sham+cold stress (SC group); myocardial ischemia+normal temperature (I group); myocardial ischemia+cold stress (IC group); atorvastatin treatment group (A group). Co-stress