EFFECTS OF ATORVASTATIN ON EXPRESSION OF MYOCARDIUM HEAT SHOCK PROTEIN 70mRNA AND INDUCEMENT OF ATRIAL FIBRILLATION INDUCED BY RAPID ATRIAL PACING IN RABBIT

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10.1136/heartjnl-2011-300867.214

Background Increasing evidence suggests that tatin is more effective in preventing paroxysmal AF than angiotensin-converting enzyme inhibitor and B-block agents. The presumed mechanism is that tatin might increase the level of Myocardium Heat Shock Protein 70 mRNA in atrial. In this study, we investigate the effects of atorvastatin pre-disposed on atrium of electrical remodeling and the expression of HSP70mRNA in rabbits with atrial fibrillation induced by rapid atrial pacing.

Methods Twenty-four healthy adult New Zealand rabbits were divided into Pacing group (n=8) heat shock+pacing group, and atorvastatin+pacing group (n=8). The hearts were paced with 2 to 4 voltage at 600 bpm frequency for 6 h. Atorvastatin was given 2.0 mg/kg/d by gavage one week before pacing. The inducibility of AF, atrial effective refractory period, adaptation of AERP were measured at 0h, 2h, 4h, and 6 h after pacing. HSP70mRNA, Cav1.2, α1c and KCa3.1mRNA of atrial tissues were detected by RT-PCR.

Results (1) The inducibility of AF was 37.5%, 75%, 100% and 100% in pacing group; 7.5%, 50%, 50% and 50% in heat shock+pacing group, and 37.5%, 50%, 50%, 62.5% in atorvastatin+pacing group at 0h, 2h, 4h, 6h after pacing. The level of HSP70 mRNA was not changed significantly, while the AERP was shortened, the adaptation of AERP was decreased as well as Cav1.2, α1c and KCa3.1 mRNA were reduced in atrial tissues in pacing group. (2) The level of HSP70 mRNA was increased significantly, the AERP, the adaptation of AERP as well as Cav1.2, α1c and KCa3.1 mRNA of atrial tissues were not changed significantly as pacing group in both heat shock+pacing and atorvastatin+pacing groups at 0h, 2h, 4h, 6h after pacing.

Conclusion We observed that Atorvastatin increased the level of HSP70 mRNA, prevented atrial electrical remodeling and reduced AF. Thus, our study suggests that stabilisation of AERP, adaptation of AERP as well as Cav1.2, α1c and KCa3.1 mRNA at atrial tissues, mediated through HSP70, is the possible mechanism of Atorvastatin in decreasing AF.