EFFECTS OF ATORVASTATIN ON TRANSIENT SODIUM CURRENTS IN RAT NORMAL/SIMULATED ISCHEMIA/REPERFUSION VENTRICULAR CELL

Bian Bo, Wan Zhen, Li Hongshi, Wang Fang, Teng Tianmin
Tianjin Medical University General Hospital, Tianjin, China

10.1136/heartjnl-2011-300867.61

Background Some clinical trials have shown statins have anti-arrhythmic effects and can improve clinical results. But its mechanism is unclear.

Objective observing the effects of atorvastatin on transient sodium currents in rat normal/simulated ischemia/reperfusion ventricular cell.
Abstracts

Methods Taking whole-cell patch clamp method to record $I_{Na}$ and measuring the expression level of SCN5A by western blot technique of simulated ventricular ischemia/reperfusion cell.

Results The short-time effects of atorvastatin on the rat normal and simulated ischemia ventricular peak $I_{Na}$ were inhibited about 25% ($p<0.05$), and after elution, inhibition disappeared. However 15 min after simulated ischemia atorvastatin inhibited the $I_{Na}$ decreasing progress. In simulated reperfusion status, $I_{Na}$ reduced and atorvastatin inhibited the reduction degree, while $I_{Na}$ of the atorvastatin and wortmannin combination group had no difference with which of reperfusion group ($p>0.05$). The expression level of SCN5A had the almost same changes with $I_{Na}$.

Conclusion (1) The short time (3 min) effect of Atorvastatin in $I_{Na}$ of the normal and simulated ischemia rat ventricular myocytes is inhibition, similar to sodium channel blockers. (2) Atorvastatin can protect the decrease of $I_{Na}$ in the status of simulated long-time (>15 min) ischemia/reperfusion. (3) Effects of Atorvastatin in the status of simulated ischemic/reperfusion can be partly overcome by Wortmannin, which means atorvastatin can affect $I_{Na}$ and the expression level of SCN5A through the way of RISK signal pathway especially of PI3K.