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EXPRESSION OF HSP70 MRNAS DURING ISCHEMIA-REPERFUSION IN THE RAT BLADDER

Aims of Study

Ischemia-reperfusion injury in the bladder is often observed during acute/chronic urinary retention and following decompression.[1-5] The main etiology of this injury is thought to be caused by free radicals, which attack cell membrane, proteins, RNAs, and DNAs.[1-5] For the process of recovery from this injury, the knowledge of bladder alteration and dysfunction after rescue of the urinary retention patients is important. Heat shock protein (HSP) 70-1/2 play an important role to repair damaged tissue after ischemia-reperfusion injury.[6] In this study, we attempted to investigate the expression of HSP 70-1/2 mRNAs, and HSP 70 proteins during ischemia-reperfusion in the rat bladder.

Methods

Rat abdominal aorta were clamped with a small clip to induce ischemia-reperfusion injury in the rat bladder dome.[1-5] Male Wistar rats, 8 weeks old, were divided into six groups: controls , 30 min ischemia, 30 min ischemia and 30, and 60 minutes, 1 day, and 1 week reperfusion, groups A, B, C, D, E, F respectively. In functional studies, contractile responses to carbachol and 100 mM KCl were measured in these groups. HSP 70-1/2 mRNAs in the experimental bladder were measured by real time PCR methods. The expressions of HSP 70-1/2 mRNAs were quantified according to compared the expression of beta actin mRNAs in the experimental rat bladders. Furthermore, the expression of HSP-70 proteins was measured using StressXpress HSP 70 ELISA kit in the bladders (Stressgen Biotechnologies, Victoria, BC, Canada).

Results

Experimental data in this study is shown in the TABLE. In functional study, Emax values of carbachol to bladders in A, B, C, D, E and F groups were 9.3 ± 1.3, 7.9 ± 1.7, 4.3 ± 0.8, 4.2 ± 0.7, 4.5 ± 0.6 and 8.1 ± 1.2 g/mm^2, respectively. Contractile response to 100mM KCl showed the same manner as Emax values of carbachol. In the control group, expressions of HSP 70-1/2 mRNA were detected. Expressions of HSP 70-1 and 2 mRNAs increased in groups B and C, which were decreased in groups D, E and F. The expression of HSP 70 proteins was increased after a short interval period of the expression of their mRNAs.

Conclusions

1) ischemia induced by clamping of the rat abdominal aorta reduces the bladder dome's contractile responses to carbachol, and subsequent reperfusion causes additional damage to smooth muscle. 2) ischemia-reperfusion injury is partially recovered after 7-day reperfusion. 3) the peak expressions of HSP 70-1/2 mRNAs in the bladder is reached 30 minutes reperfusion after the insult of 30 minutes of ischemia. 4) The expression of HSP 70 proteins is increased after a short interval period of the expression of their mRNAs

TABLE EXPRESSION OF HSP 70-1/2 mRNAS IN THE RAT BLADDER

Groups	HSP 70-1/beta actin	HSP 70-2/beta actin
A	3.26 ± 0.57	0.74 ± 0.14
B	9.12 ± 3.15*	0.94 ± 0.47
C	5.08 ± 0.85	1.41 ± 0.16**
D	5.85 ± 1.44	0.40 ± 0.11
E	4.33 ± 1.52	0.73 ± 0.26
F	3.53 ± 0.82	0.33 ± 0.15

*) significantly different from group A. **) significantly different from groups A, E and F.

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