

PREVENTIVE EFFECT OF LONG CHAIN FATTY ALCOHOL ON ISCHEMIA-REPERFUSION INJURY IN THE RAT BLADDER

Aims of Study

There is an increasing evidence that free radicals caused by ischemia-reperfusion induce bladder dysfunction in acute and chronic urinary retention, and subsequent catheterization.¹ If this hypothesis is correct, one of the most important issue to prevent bladder dysfunction in BPH patients is to reduce the production of free radicals in the bladder. In this study, we attempted to clarify the preventive effects of cyclohexenonic long-chain fatty alcohol (FA), which is reported to be a kind of scavengers, on ischemia-reperfusion injury in the rat bladder.² Furthermore, we attempted to measure malonaldehyde (MDA) as makers of lipid peroxidation in the bladder.¹

Methods

Eight weeks male Wistar rats were used in this study. Rat abdominal aorta was clamped with a small clip to induce ischemia-reperfusion injury in the rat bladder dome. Clamping of the abdominal aorta (ischemia) decreased the blood flow in the rat urinary bladder to 5%-10% of the preclamping level. Since our preliminary experiments revealed that 30 minutes ischemia did not cause significant bladder dysfunction and that 30 minutes reperfusion caused more severe damage than 5, 10 or 20 minutes reperfusion, rats in this study were exposed to 30 minutes ischemia-30 minutes reperfusion. The rat abdominal aorta was clamped to induce ischemia-reperfusion (I-R) injury in the rat bladder with or without FA (0.5, 2, 8 mg/kg, i.p. 30 minutes prior to the ischemia). Muscle bath studies to carbachol and 100 mM KCl were performed to confirm the bladder function. MDA concentrations in the tissue were measured by colorimetric assay in these groups. Statistical analysis of the differences between groups was performed using analysis of variance and the multiple comparison Fisher's test. $P \leq 0.05$ was regarded as the level of significance.

Results

Bladder dysfunction caused by ischemia-reperfusion is prevented by FA in dose dependent manner. Emax values of bladder strips to carbachol from control, 30 minutes ischemia-30 minutes reperfusion without FA, with 0.5, 2, 8 mg/kg FA groups were 9.0 ± 0.9 , 3.2 ± 0.4 , 4.5 ± 0.5 , 5.4 ± 0.4 , 6.5 ± 0.5 g/mm², respectively. Contractile response induced by 100 mM KCl was similar to Emax value response to carbachol in all groups. MDA concentrations in the bladder tissue from control, 30 minutes ischemia-30 minutes reperfusion without FA, with 0.5, 2, 8 mg/kg FA groups were 6.1 ± 1.4 , 14.2 ± 2.5 , 10.1 ± 1.0 , 8.1 ± 1.9 , 6.5 ± 0.9 microM/g Tissue, respectively. Lipid peroxidation in the experimental bladder was prevented by treatment with FA in dose dependent manner.

Conclusions

1) Ischemia induced by clamping of the rat abdominal aorta caused reduction in contractile responses to carbachol of the bladder dome, and subsequent reperfusion caused additional damage to smooth muscle judged by functional study; 2) Lipid peroxidation on ischemia-reperfusion injury can be prevented by treatment with radical scavengers; 3) Ischemia reperfusion injury was prevented by treatment with FA.

References

1. Saito, M. and Miyagawa, I.: Bladder dysfunction after acute urinary retention in rats. *J. Urol.* 165: 1745-1747, 2001.
2. Watanabe, T. and Miyagawa, I.: Effect of long chain fatty alcohol on peripheral nerve conduction and bladder function in diabetic rats. *J. Urol* 165:275, 2001