

BIOCHEMICAL INTERPRETATION OF ISCHEMIA-REPERFUSION INJURY AFTER ACUTE URINARY RETENTION IN RAT BLADDER

Hypothesis / aims of study

In Benign Prostatic Hyperplasia (BPH), acute urinary retention results in ischemia by decreasing bladder blood flow. The blood flow increases following catheterization and tissue injury occurs by reactive oxygen species (ROS) which is a product of ischemia-reperfusion of bladder [1.2]. The aim of this study was to see the effects of ischemia-reperfusion injury on ROS in acute urinary retention and to determine whether these effects would be normalized by different antioxidant agents. Another aim of this study was to investigate the extra contribution of recurrent urinary retention on this event.

Study design, materials and methods

We used 45 male Wistar Albino rats in our research. We formed six groups totally, as a combination of 1 sham, 2 retention and 3 treatment groups including 5, 8, 8 male rats in each group respectively. In the treatment groups allopurinol and/or verapamil were used. In retention group, Following urethral catheterisation the rat penile urethra was clamped, diuresis was forced and overdistension was maintained. After 30 minutes under overdistension the rats bladder was emptied with 3F catheter. After reperfusion of their bladders and waiting for 30 minutes in this condition, the rats were sacrificed. The same procedure was administered again one week later before sacrifice in recurrent retention group. In the treatment groups allopurinol and/or verapamil was administered intraperitoneally before second catheterisation. Bladder tissue samples were taken and malondialdehyde(MDA)-myeloperoxidase(MPO) measurements were made.

Results

The results were shown at the table.

Group	Mean MDA \pm SD (nmol/gr tissue)	Mean MPO \pm SD (U/gr tissue)
Sham	8.70 \pm 8.41	0.38 \pm 0.18
Retention	29.34 \pm 13.54	0.89 \pm 0.39
Recurrent retention	24.42 \pm 7.89	0.84 \pm 0.33
Allopurinol	14.00 \pm 9.54	0.52 \pm 0.18
Verapamil	14.22 \pm 11.89	0.53 \pm 0.21
Allopurinol+Verapamil	9.23 \pm 2.62	0.44 \pm 0.12

Interpretation of results

The MDA – MPO increase in the retention groups show that the decompression following acute urinary retention, leads ischemia-reperfusion injury in the bladder. Leukocytes have a role in this injury. In the retention groups both MDA and MPO levels were increased significantly when compared with the sham group. The increase in these parameters in the recurrent retention group wasn't statistically different from the retention group. In the treatment groups, decrease in MDA and MPO levels were found statistically important when compared with the retention group. We didn't find any significant difference within the treatment groups and also between treatment groups and sham group. We didn't find any additional biochemical injury in the recurrent retention group. We found that both allopurinol and verapamil were effective in decreasing the injury and the combination of these agents, compared with the mono-treatment, didn't make any additional benefit.

Concluding message

In conclusion, decompression in acute urinary retention leads ischemia-reperfusion injury. Recurrent urinary retention doesn' t add any further reperfusion injury and treatment with antioxidants prevents this injury. But biochemical MDA and MPO parameters are not enough to make a clinical comment about the role of ischemia-reperfusion injury on bladder. Our results should be supported with histopathological interpretation and contractility studies.

References

- 1- Bladder dysfunction after acute urinary retention in rats. J Urol 2001; 165: 1745-1747.
- 2- Duration of increased mucosal permeability of the urinary bladder after acute overdistension: an experimental study in rats. U Res 1999; 27: 272-6.