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EFFECTS OF L-ARGININE ON BLADDER FUNCTION IN BLADDER-OUTLET-OBSTRUCTED RATS

Aims of Study

Bladder outlet obstruction (BOO) is known to cause bladder dysfunction. Although the mechanism underlying BOO is still obscure, BOO is reported to increase intravesical pressure and thereby to decrease blood flow in the bladder wall, resulting eventually in bladder dysfunction[1]. Nitric oxide (NO) mediates relaxation of smooth muscle and plays a major role in regulating blood flow [3]. Although there have been no reports on the measurement of NO in the obstructed bladder, it is speculated that an increase of NO in the detrusor prevents BOO-induced bladder dysfunction. The present study investigates whether or not L-arginine, a precursor of NO synthesis, prevents BOO-induced bladder dysfunction.

Methods

Thirty-six 12-week-old female Wistar rats were divided into sham group, BOO group and L-arginine group. Each of which was examined at 1 week or 6 weeks after treatment and there was two sham groups, which received sham operations; two BOO groups, in which BOO was surgically induced according to Mattiasson and Uvelius [2]; and two L-arginine groups, in which L-arginie was injected intra-peritoneally for 1 week or for 6 weeks after surgical induction of BOO. After treatment was completed, micturition behavior was monitored, and a cystometrogram under urethane anesthesia was performed. Immediately after the cystometrogram, the bladder was removed, weighed, and used for the strip study, measurement of NOS activity, and histological examination. In the strip study, contractile responses to carbachol and KCL were monitored, and in the histological study, the ratio of area density of smooth muscle versus connective tissue was determined.

Results

For the BOO rats, the bladder weight, the number of micturitions, the maximum detrusor pressure during voiding, the residual urine volume, and the ratio of the area density of smooth muscle to connective tissue were significantly higher than those of the sham-operated rats. On the other hand, no increase over sham groups were seen in the rats of the L-arginine groups. In NOS activity, there was no significant difference among groups. In the contractile responses to carbachol and KCL, the BOO groups showed significantly lower Emax and ED50 values than did the sham-operated groups, and the L-arginine groups significantly recovered compared to the BOO groups.

Conclusions

L-arginine improved bladder dysfunction in the BOO rats. This may be attributed to L-arginine increasing NO levels and blood-flow rate in the obstructed bladder. In addition, NO may play an important role in preventing the bladder damage induced by BOO.

RERERENCES

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