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URINARY EXCRETION OF GLYCOSAMINOGLYCANS (GAGS) IN PATIENTS WITH OVERACTIVE DETRUSOR CONTRACTIONS : PRELIMINARY REPORTS

Aims of Study

Its well known that glycosaminoglycans (GAGs) are the major components of the connectivum and that they are located also on the luminal surface of the bladder (1). Studies in humans and rodents have shown that in normal bladder glycosaminoglycans act as a permeability barrier controlling the movement of charged molecules. Recently some authors have studied the possible role of GAGs in nocturnal enuresis determining variation of amount of GAGs excreted in urine of young patients with this pathology (2) but without any correlation between urodynamic findings and urinary excretion of GAGs . Whether urinary excretion of GAGs is related to a detrusor overactivity is still unknown.
The aim of the present study was to investigate variantions in GAGs excretion in patients with overactive detrusor contraction (ODC) to ascertain the possible role of GAGs as marker of this pathology.

Methods

The study included sixteen patients (7 women and 9 men, mean age of 63.81 years, range 40-79) presenting urgency no treated by anticholinergic drugs. No patient had urinary tract infection (UTI) or abnormally renal function. These patients underwent to urodynamic study and all of them showed a pattern of detrusor overactivity. In all patients the following urodinamic parameters were considered : a) ODC (overactive detrusor contraction) threshold, expressed as the bladder filling volume at which first ODC start; b) the ODC amplitude, which represents the maximal detrusor pressure recorded during ODC; c) the ODC duration as the mean, for each patient, in seconds from the beginning to the end of ODC; d) the bladder capacity as the bladder volume at which urine leakage was observed or the bladder volume at the unbearable desire to void. The study also included 9 healthy controls (2 women and 7 men, mean age of 65.75 years, range 23-79). The urinary excretion of GAGs was measured in all patients and controls after collecting 24-h urine samples. The creatinine concentration was also measured in the urine collected from each patient. The total GAG content was expressed as a ratio of the creatinine content and a Mann-Whitney test was employed to verify any statistical difference between the urinary excretion of GAGs in pathological group and the urinary excretion of GAGs in control group ($P < .05$ was considered significant).

Results

The urodynamics showed a mean ODC threshold of 108.19 ml (mean SE 22.91/ SD 91.64), a mean ODC amplitude of 57.88 cm H2O (mean SE 9.61/ SD 38.46), a mean ODC duration of 48.63 sec (mean SE19.53/SD 78.10) and a mean bladder capacity of 256.25 ml (mean SE 21.90/SD 87.60). In particular the statistical analysis revealed a significant higher mean urinary content of GAGs in the control group than in the pathological group in relation both to the ratio GAGs/urine ($P = .043$) and to the ratio GAGs/creatinine ($P = .015$) (tab.1).

	GAGs/urine (µg/ml)	GAGs/creatinine (mg/g)
Control group	mean : 0.84 mean SE : 0.22 SD : 0.66	mean : 2.03 mean SE : 0.61 SD : 1.82
Pathological group	mean : 0.48 mean SE : 0.20 SD : 0.79	mean : 0.57 mean SE : 0.16 SD : 0.61

Tab.1

Conclusions

In the present study we observed unexpectedly a low urinary excretion of GAGs in pathological group in comparison with control group. The very high amplitude and duration of ODC suggested that the low excretion of GAGs in the pathological group could be the result of a chronic stressful and strong mechanical activity of the detrusor muscle.

References

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