117# CLINICAL VALUE OF URINARY EXCRETION OF PROSTAGLANDINS E_2, $F_{2\alpha}$ AND C-REACTIVE PROTEIN (CRP) IN WOMEN WITH IDIOPATHIC DETRUSOR OVERACTIVITY

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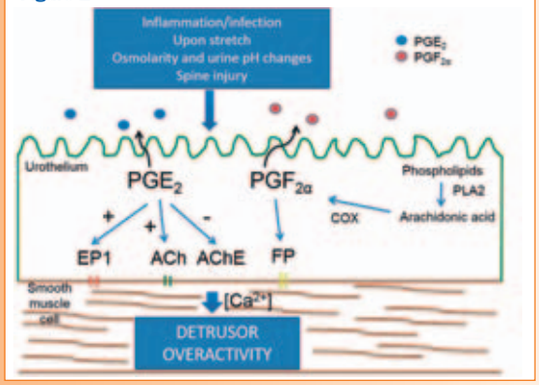
Hypothesis/aims of study

Arachidonic metabolites, more specifically PGs, are released from the bladder into the general circulation in response to distension. It was found that PGs in the bladder wall originate from both the urothelial and muscle layers. The exact role of this endogenous PG is not known, but it is well documented that exogenous PG alters bladder motor activity in vitro and in vivo and that it can also influence the micturition reflex in humans, rats, guinea pigs, rabbits and monkeys. The main PGs synthesized in the bladder are PGE_2 and PGI_2. PGs are locally synthesized in the bladder muscle and mucosa. This synthesis is initiated by stretch of the detrusor muscle, bladder nerve stimulation, bladder mucosa damage and inflammation mediators (*Figure 1*).

The purpose of this study was to determine the urinary excretion of the prostaglandins E_2 and $F_{2\alpha}$ and PCR in women with idiopathic detrusor overactivity in order to identify the potential value of these substances as a noninvasive method in evaluating women with symptoms of overactive bladder.

Figure 1



Study design, materials and methods

Prospective study with a total of 43 women with urodynamic diagnosis of idiopathic detrusor overactivity and a control group of 31 patients without OAB symptoms and a normal urodynamic study. The mean age of the patients in the study group was 60 years (range 25-81) and in the control group of 58 years (range 34-80). Urine samples from 74 patients were collected and determined by ELISA PGE_2 levels (Arbor Assays®), $PGF_{2\alpha}$ (Enzo Life Sciences®) and PCR. The results were normalized by the urinary creatinine concentration. Levels of marker/Cr between the study group (overactive detrusor) and the control group were compared. The study group was treated with solifenacin 10 mg/day. Markers were determined in urine at 30 and 90 days to evaluate the influence of treatment on the levels of prostaglandins.

Results

CRP levels detected in urine in all cases and controls were very low (<0.08 mg/L) with no differences between the two groups. Higher average levels of PGE_2/Cr (2925 vs 2632 pg/mgCr) and $PGF_{2\alpha}$/Cr (2178 vs 1837 pg/mgCr) were observed in the group of patients with overactive detrusor respect to controls, but did not reach statistical significance (p> 0,05) (*Table 1*). Treatment with solifenacin 10 mg/day significantly decreased PGE_2/Cr levels at 90 days (p=0.01). No changes were observed in the urinary levels of $PGF_{2\alpha}$ with anticholinergic therapy (*Tables 2 and 3*). (*Figures 2 and 3*).

Figure 2

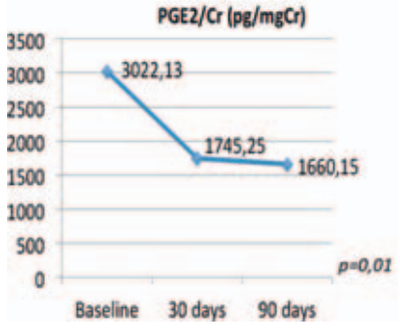


Figure 3

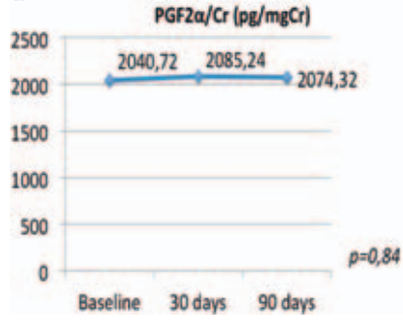


Table 1

	CONTROL GROUP (n=31)	IDO PATIENTS (n=43)	P
PGE2 (pg/mgCr)	2632,29 ± 2101,38	2925,92 ± 2813,05	0,62
PGF2α(ng/mgCr)	1837,90 ± 1216,84	2178,64 ± 1635,42	0,30
PCR (mg/L)	<0,08	<0,08	>0,05

Table 2

	Baseline	Solifenacin 10mg/dy x 30dys	P	n
PGE_2 (pg/mgCr)	3022,13 ± 2861,02	1745,25 ± 2776,55	0,07	33
$PGF_{2\alpha}$ (pg/mgCr)	2040,72 ± 1438,14	2085,24 ± 1500,49	0,84	33

Table 3

	Baseline	Solifenacin 10mg/dy x 90dys	P	n
PGE_2 (pg/mgCr)	3045,41 ± 2991,72	1660,15 ± 1232,72	0,01	25
$PGF_{2\alpha}$ (pg/mgCr)	2056,66 ± 1584,76	2074,32 ± 1192,70	0,95	25

Interpretation of results

Treatment with solifenacin 10 mg/day significantly decreases the levels of PGE_2 in the urine of patients with IDO, so this molecule could be involved in the pathophysiology of overactive bladder and may be useful as a biomarker in monitoring response to treatment. The PCR and $PGF_{2\alpha}$ have not demonstrated clinical value for this condition.

Concluding message

According to our results, PGE_2 may have a role in monitoring response to anticolinergic treatment, but more studies with a larger number of patients are needed to confirm this observation.

Disclosures

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