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IS BLADDER ISCHEMIA A CAUSE OF LUTS? OBSERVATIONS IN PATIENTS WITH MARKED PELVIC ARTERIAL HYPOPERFUSION

Hypothesis / aims of study

Animal models of iliac arterial occlusion (IAO) showed that chronic bladder ischemia can affect bladder function.[1,2] On the other hand, clinical studies investigated possible associations between atherosclerosis risk factors and LUTS, but the relationship between pelvic arterial hypoperfusion (PAH) and LUTS was never explored.

Herewith, we investigated LUTS and urinary levels of neurotrophins in patients with marked PAH in order to validate the experimental model of IAO.

Study design, materials and methods

Twenty-seven male patients from the vascular surgery clinic were enrolled.

Fourteen patients had PAH and were categorized according to its severity: 9 had unilateral internal iliac occlusion (UIIO) and 5 had bilateral internal iliac occlusion (BIIO).

The control group (n=13) included patients submitted to carotid endarterectomy, without evidence of aortoiliac occlusive disease.

Exclusion criteria included neurogenic bladder dysfunction, bladder or prostate cancer, pelvic radiotherapy or chronic treatment for LUTS.

All patients were evaluated clinically (Rutherford classification) and anatomically (aortoiliac TASC II classification) for peripheral artery disease, using angio-CT scan or angiography.

Participants underwent urological examination, including IPSS score, uroflowmetry, postvoid residual (PVR) and prostate volume determination.

Urine samples were collected from all participants and ELISA measurement of NGF and BDNF was performed.

Results

Data are summarized in the table. Subjects with PAH had mild LUTS and although the mean IPSS score was higher than in controls, the difference was not statistically significant (9.8±4.6 vs. 7.9±1.7, p=0.26). Qmax and PVR were similar in both groups. There was a trend for patients with more severe PAH (BIIO) to have smaller prostate volumes (p=0.07).

Patients with PAH had significant higher urinary levels of NGF in comparison to controls (3.59±0.73 vs 2.9±0.72, p=0.05). This did not occur for BDNF.

	Controls	**Pelvic Arterial Hypoperfusion (vs. controls)**			
		UIIO (vs. controls)	P	**BIIO (vs. controls)**	P
N	13	9		5	
Age	68.9±8.0	72.2±9.2	>0.05	61.8±4.0	>0.05
TASC II classification (grade C/D) (%; n)	8 (1)	25 (2)	>0.05	**60 (3) ***	**<0.001**
Rutherford classification	0.5±1.1	1.0±1.8	>0.05	**3.8±1.1 ***	**<0.001**
IPSS	7.9±1.7	9.8±4.6 (p>0.05)			
		10.8±3.0	>0.05	8.4±4.3	>0.05
PVR (ml)	38.4±28.7	29.2±25.4			
		27.2±23.7	>0.05	31.6±29.1	>0.05
Qmax (ml/s)	16.9±8.0	14.5±4.6 (p>0.05)			
		13.7±3.5	>0.05	15.8±6.3	>0.05
Prostate volume (ml)	37.8±20.8	30.7±17.1 (p>0.05)			
		35.6±19.2	>0.05	**22.8±10.1 ***	**0.07**
NGF/Creatinine (log)	2.93±0.72	**3.59±0.73 (p=0.05) ***			
		3.71±0.96	0.11	3.44±0.40	0.14
BDNF/Creatinine (log)	3.19±0.52	3.34±0.64 (p=0.66)			
		3.55±0.32	0.87	3.21±0.84	0.83

Data is expressed as the mean ± standard deviation
TASC II classification – anatomical classification of aortoiliac disease
C and D categories indicate more severe anatomical disease
Rutherford classification – clinical classification of peripheral arterial disease
Higher score indicates more severe symptomatic disease
* p <0.05 is statistically significant

Interpretation of results

In this pilot study, chronic moderate to severe PAH was not associated with severe LUTS, suggesting that, in man, the eventual development of collateral irrigation of the bladder might delay or prevent significant bladder dysfunction. The low-grade inflammation associated with ischemia can explain the marginal increase of urinary NGF.

Concluding message

This study seems to limit the human translation of the findings obtained in animal models of chronic bladder ischemia induced by IAO.

References

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Disclosures

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