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IS SYSTEMIC VASCULAR ENDOTHELIAL DYSFUNCTION RELATED TO BLADDER OUTLET OBSTRUCTION IN THE MALE? A PRELIMINARY STUDY

Hypothesis / aims of study
It is well recognized that endothelial dysfunction is strongly related to lower urogenital problems such as erectile dysfunction, which shortly precedes clinical coronary arterial occlusion. The study aims to investigate the potential correlation of systemic vascular endothelial dysfunction and coronary artery disease (CAD) with bladder outlet obstruction (BOO).

Study design, materials and methods
Thirty men age 50 years and older (mean=62.7 years ±14.6) with moderate to severe LUTS were enrolled and gave written consent. All patients underwent pressure flow study and simultaneous non-invasive recording of detrusor tissue oxygenation and haemodynamics with Near Infrared Spectroscopy (NIRS). Systemic endothelial integrity was assessed by measurement of inflammatory and endothelial-prothrombotic markers such as Endothelin-1 (ET-1), Interleukin-6 (IL-6), Assymetric Dimethylarginine (ADMA) and N-terminal C-type natriuretic peptide (NT-pro CNP). CAD, defined as occlusion of coronary arteries, was also recorded. IBM-SPSS ver. 22 was used for the statistical analysis.

Results
Logistic regression model was applied to examine whether BOO – as diagnosed properly with the standard Pressure Flow study and also by estimation of detrusor muscle oxygenation with the NIRS technique – correlates with vascular lesions. Elevated values of ET-1, IL-6 and NT-proCNP were found, but they were not statistically significant. NIRS showed a specificity of 88% and a sensitivity of 79% in diagnosing BOO. Also, a sevenfold increase was found in the likelihood of BOO in patients with CAD.

Interpretation of results
Elevated biochemical markers of endothelial dysfunction such as ET-1, IL-6 and NT-proCNP, as well as by the sevenfold increase in the likelihood of BOO in coronary patients suggest that there may be a correlation between lower urinary tract dysfunction and vascular occlusion. With the limitation of the small sample size, a common pathophysiological background may exist such as impaired blood perfusion of the bladder and/or prostate. This is also supported by the use of NIRS that assesses obstruction through detrusor oxygenation impairment.

Concluding message
Bladder outlet obstruction may be related to endothelial dysfunction and coronary artery disease. Also, NIRS may be a good alternative for non-invasive urodynamic studies in the assessment of bladder outlet obstruction.

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Disclosures
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