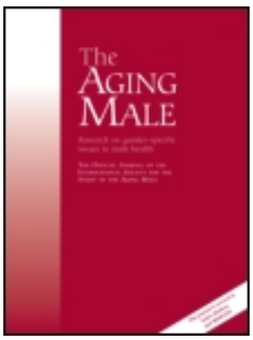

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EDITORIAL

Benign prostatic hyperplasia and metabolic syndrome: the expanding evidences of a new disease of aging male

Metabolic syndrome (MetS) is a complex and widespread epidemic disorder with a high socio-economic impact, due to its association with increased morbidity and mortality. A significant amount of epidemiological evidence have underlined the emerging link between MetS, benign prostatic enlargement secondary to benign prostatic hyperplasia (BPH) and related lower urinary tract symptoms (LUTS) [1–3].

Among these features, the presence of hyperinsulinemia has been taken into account as the most important key factor. Hyperinsulinemia is in fact associated with an increase in the activity of the sympathetic nervous system and this may contribute to the increased muscle tone of the prostate, resulting in more severe LUTS independently from prostate enlargement [4]. In fact, accumulating evidence indicates that glucose homeostasis, hyperinsulinemia, and insulin resistance may increase the risk of BPH [5–7].

Among MetS features, reduced HDL and increased triglyceride levels were significantly related to higher prostatic inflammation by secreting IL-8 in response not only to oxidated LDL, but also to insulin [8], indicating that different MetS features could synergistically boost inflammation and tissue-remodeling in BPH/LUTS [9,10].

In addition to age, LUTS and MetS also share a variety of other risk factors, such as obesity, high fasting plasma glucose levels, hypertension, androgen deficiency, depression, and smoking, thus indicating that metabolic syndrome might play a key role in the pathogenesis of LUTS [11]. Taken into account these premises, it should be noted that all previous pathogenetic mechanisms are known to increase cardiovascular disease (CVD) risk [12].

In a recent cross-sectional study in a cohort of patients with BPH/LUTS, it has been demonstrated an increase of more than 5-fold of having a Framingham risk score $\geq 10\%$ in men with moderate–severe LUTS [13].

Although a direct link between LUTS/BPH occurrence and increased CVD-risk cannot be directed linked, it may be postulated as common pathogenic pathways between both diseases. In this pathological circle, non-alcoholic fatty liver disease (NAFLD) has been proposed as the hepatic manifestation of the MetS, with IR as the common path physiological factor.

Insulin resistance is the underlying mechanism of metabolic syndrome and is also the most prevalent risk factor for NAFLD, a condition strongly associated with hepatic and adipose tissue insulin resistance, as well as reduced whole-body insulin sensitivity

Interestingly, MetS induced inflammation within the liver, also known as steato-hepatitis, has been very recently elucidated in an experimental study in the rabbit [14]. Surprisingly, a recent cross-sectional study showed a new link between the presence of BPH/LUTS, MetS and NAFLD, demonstrating a 2.0-fold risk of having moderate–severe LUTS in men with both manifestation ($OR = 2.10$, $p < 0.01$) [15]. All these findings suggest that the MetS-related BPH/LUTS should be considered as potentially targeted therapy in order to counteract the resulting prostate overgrowth.

BPH/LUTS is often considered as a normal consequence of the aging men leading to medical and therefore surgical intervention [16]. We would breakdown this view and unveil that the progression of this condition could be prevented by modifying previous metabolic factors. In our point of view, BPH/LUTS may be considered as a complex disorder that can also be discovered in the earlier stage.

Primum non- ‘‘nocere’’ (first do no harm) should always be kept in mind, and therefore the counteraction of previous metabolic alterations should be the milestone of BPH/LUTS treatment. The next challenges of urologists should be the contrast of early onset of BPH/LUTS and the development of new target therapies in men at risk.

Declaration of interest

The authors report no declarations of interest.

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References

1. Yeh HC, Liu CC, Lee YC, et al. Associations of the lower urinary tract symptoms with the lifestyle, prostate volume, and metabolic syndrome in the elderly males. Aging Male 2012;15:166–72.

2. Aktas BK, Gokkaya CS, Bulut S, et al. Impact of metabolic syndrome on erectile dysfunction and lower urinary tract symptoms in benign prostatic hyperplasia patients. Aging Male 2011;14:48–52.
3. Demir O, Akgul K, Akar Z, et al. Association between severity of lower urinary tract symptoms, erectile dysfunction and metabolic syndrome. Aging Male 2009;12:29–34.
4. Russo GI, Cimino S, Fragala E, et al. Insulin resistance is an independent predictor of severe lower urinary tract symptoms and of erectile dysfunction: results from a cross-sectional study. J Sex Med 2014;11:2074–82.
5. Gacci M, Eardley I, Giuliano F, et al. Critical analysis of the relationship between sexual dysfunctions and lower urinary tract symptoms due to benign prostatic hyperplasia. Eur Urol 2011;60: 809–25.
6. He Q, Wang H, Yue Z, et al. Waist circumference and risk of lower urinary tract symptoms: a meta-analysis. Aging Male 2014;17: 223–9.
7. Ferreira FT, Daltoe L, Succi G, et al. Relation between glycemic levels and low tract urinary symptoms in elderly. Aging Male 2015; 18:34–7.
8. Gacci M, Vignozzi L, Sebastianelli A, et al. Metabolic syndrome and lower urinary tract symptoms: the role of inflammation. Prostate Cancer Prostatic Dis 2013;16:101–6.
9. Vignozzi L, Gacci M, Cellai I, et al. PDE5 inhibitors blunt inflammation in human BPH: a potential mechanism of action for PDE5 inhibitors in LUTS. Prostate 2013;73:1391–402.
10. Vanella L, Russo GI, Cimino S, et al. Correlation between lipid profile and heme oxygenase system in patients with benign prostatic hyperplasia. Urology 2014;83:1444 e7–13.
11. Fusco F, D'Anzeo G, Sessa A, et al. BPH/LUTS and ED: common pharmacological pathways for a common treatment. J Sex Med 2013;10:2382–93.
12. Bouwman, II, Blanker MH, Schouten BW, et al. Are lower urinary tract symptoms associated with cardiovascular disease in the Dutch general population? Results from the Krimpen study. World J Urol 2014. [Epub ahead of print]. doi: 10.1007/s00345-014-1398-y.
13. Russo GI, Castelli T, Privitera S, et al. Increase of Framingham risk score is associated with severity of Lower urinary tract symptoms. BJU Int 2015. [Epub ahead of print]. doi: 10.1111/bju.13053.
14. Vignozzi L, Filippi S, Comeglio P, et al. Nonalcoholic steatohepatitis as a novel player in metabolic syndrome-induced erectile dysfunction: an experimental study in the rabbit. Mol Cell Endocrinol 2014;384:143–54.
15. Russo GI, Cimino S, Fragala E, et al. Relationship between non-alcoholic fatty liver disease and benign prostatic hyperplasia/lower urinary tract symptoms: new insights from an Italian cross-sectional study. World J Urol 2014. [Epub ahead of print]. doi: 10.1007/s00345-014-1392-4.
16. Favilla V, Cimino S, Salamone E, et al. Risk factors of sexual dysfunction after transurethral resection of the prostate (TURP): a 12 months follow-up. J Endocrinol Investig 2013;36:1094–8.