Letter

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Letter to the editor: Impact of metabolic syndrome on response to medical treatment of benign prostatic hyperplasia

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We have read the article by Cyrus et al. [1], which was an interesting study investigating the impact of metabolic syndrome (MetS) on the treatment of benign prostatic hyperplasia (BPH). In their study, Cyrus et al. tried to evaluate the effect of MetS on the response to combination therapy with an α-blocker and 5α-reductase inhibitor (5ARI) after a 3-month period of therapy. Although the mean prostate volume of the patients with MetS was significantly higher than that that of the controls, significant differences in the International Prostate Symptom Score and its components were shown between patients with and without MetS. The authors suggested with this finding that MetS negatively affected the clinical response of BPH to medical treatment.

Hyperinsulinemia in patients with BPH might have increased sympathetic nervous system activity, which contributes to an increase of prostate smooth muscle tone [2]. Selective α1-blockers reduce urethral closure resistance and inhibit smooth muscle tone in the prostate and are the first-line medical treatment option for men with BPH. 5ARIs block the conversion of testosterone to dihydrotestosterone and are approved for the treatment of BPH.

The duration of treatment with finasteride in the study was 3 months. In the European Association of Urology Guidelines, which include benign prostatic obstruction, it has been indicated that the clinical efficacy of 5ARI relative to placebo is seen after a minimum treatment duration of at least 6 to 12 months [3]. The effects of finasteride on prostate size have been studied extensively, with maximal reduction of prostate volume achieved within 6 months [4]. In addition to this complaint, the authors did not evaluate the effects of α-blocker and 5ARI alone in the treatment of the patients with MetS. As we mentioned above, the effects of α-blocker and 5ARI on the symptoms of patients with lower urinary tract symptoms, especially with MetS, differ.

In conclusion, the treatment of BPH in patients with MetS should differ from that in patients without MetS. The pathophysiology of BPH includes hyperinsulinemia, which contributes to an increase in prostate smooth muscle tone via increased activation of the sympathetic system, and increased estradiol levels and a decreased testosterone/estradiol balance due to increased aromatase levels. In addition, the clinical effects of 5ARIs are seen after a minimum treatment duration of at least 6 to 12 months. However, hypercortisolism, which plays an important role in the development of MetS, may arise from inhibition of 5α-reductase. Therefore, whereas the use of 5ARI improves BPH, it can be a cause of aggravation of MetS. For these reasons, patients with MetS and BPH should be treated with α-blockers as a first-line treatment.

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CONFLICTS OF INTEREST

The authors have nothing to disclose.

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