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Metabolic Syndrome, Diabetes, and BPH: Intersecting Mechanisms of Prostatic Growth

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ABSTRACT

Metabolic syndrome (MetS) and diabetes mellitus (DM) are increasingly recognized as important risk factors in the development and progression of benign prostatic hyperplasia (BPH), a common urological condition in aging men characterized by prostatic enlargement and lower urinary tract symptoms (LUTS). Although traditionally considered separate clinical entities, an expanding body of epidemiological and experimental evidence links metabolic dysfunction with prostatic growth through shared pathophysiological mechanisms including chronic systemic inflammation, hormonal alterations, insulin resistance, and oxidative stress. This review synthesizes current knowledge on how elements of MetS, particularly obesity, dyslipidemia, hypertension, and hyperglycemia, intersect with diabetes-related biochemical pathways to influence prostatic cellular proliferation and stromal remodeling. We discuss the roles of insulin/insulin-like growth factor (IGF) signaling, adipokines, sex steroids, and inflammatory mediators in modulating prostatic epithelial and stromal dynamics. Converging evidence suggests that diabetic metabolic disturbances not only increase the risk of BPH but may also contribute to faster symptom progression and reduced therapeutic responsiveness. Understanding these interconnected pathways is critical for developing more effective preventive and therapeutic strategies. We conclude by outlining potential molecular targets and clinical implications for managing BPH in men with MetS and diabetes.

Keywords: metabolic syndrome, diabetes mellitus, benign prostatic hyperplasia, inflammation, insulin resistance

INTRODUCTION

Benign prostatic hyperplasia (BPH) is a histological diagnosis defined by the proliferation of prostatic stromal and epithelial cells, leading to enlargement of the transitional zone of the prostate [1]. Clinically, BPH manifests as lower urinary tract symptoms (LUTS) that can significantly reduce quality of life. BPH prevalence increases with age, affecting up to 50% of men by age 60 and 90% by age 85 [2]. Traditional risk factors include aging and androgenic stimulation, particularly dihydrotestosterone (DHT) [3]. However, in recent decades, metabolic disorders such as metabolic syndrome (MetS) and type 2 diabetes mellitus (T2DM) have emerged as significant, modifiable contributors to the onset and severity of BPH [4]. MetS encompasses a cluster of metabolic abnormalities-central obesity, insulin resistance, dyslipidemia, and hypertension-that together increase risk for cardiovascular disease and T2DM. Epidemiological studies have consistently demonstrated a higher prevalence of BPH and LUTS in men with components of MetS and in those with overt diabetes [5]. The biological rationale for this association lies in the chronic metabolic and inflammatory milieu characteristic of MetS and DM, which may exert trophic effects on prostatic tissue [6]. This review critically examines the intersecting mechanisms by which metabolic dysfunction and diabetes influence prostatic growth, synthesizing clinical and mechanistic evidence that links systemic metabolic disturbances with local prostatic pathology.

Metabolic Syndrome and BPH: Epidemiological Links

Metabolic syndrome is defined by the presence of at least three of the following: increased waist circumference, elevated triglycerides, reduced high-density lipoprotein (HDL) cholesterol, elevated blood pressure, and elevated fasting glucose [7]. Men with MetS demonstrate a significantly higher prevalence of BPH and more severe LUTS compared with metabolically healthy peers [8]. Cross-sectional and prospective cohort studies have reported positive associations between the number of MetS components and prostate volume. Insulin resistance, a hallmark of MetS, appears particularly influential. Hyperinsulinemia promotes increased bioavailability of insulin-like growth factor 1 (IGF-1), a potent mitogen that stimulates cellular proliferation and inhibits apoptosis in many tissues, including the prostate [9]. Furthermore, dyslipidemia and hypertension contribute to vascular dysfunction and oxidative stress, which may support prostatic tissue remodeling [10]. Clinically, men with MetS often present with more severe storage and voiding symptoms, larger prostates, and a greater likelihood of acute urinary retention and surgical intervention [11]. These observations underscore the need to view BPH not simply as a localized urological condition but as part of a systemic metabolic-inflammatory disorder.

Diabetes Mellitus and Prostatic Growth

Type 2 diabetes mellitus (T2DM), characterized by chronic hyperglycemia and insulin resistance, has also been implicated in the pathogenesis of BPH [12]. Multiple studies have demonstrated that diabetic men have increased prostate volume and worse LUTS compared to non-diabetic controls. The relationship between glycemic control and BPH severity suggests that long-term hyperglycemia may exert cumulative adverse effects on prostatic tissues [13]. Mechanistically, diabetes induces a state of chronic low-grade inflammation and oxidative stress, both of which are recognized drivers of BPH [14]. Hyperglycemia enhances the production of advanced glycation end products (AGEs) and reactive oxygen species (ROS), which can activate pro-inflammatory signaling cascades in the prostate. These signals may stimulate stromal cell proliferation and extracellular matrix deposition, contributing to tissue enlargement [15]. In addition, diabetic autonomic neuropathy may impair bladder function, exacerbating LUTS independently of prostate size [16]. Therefore, the clinical picture in diabetic men with BPH often reflects a combination of obstructive and non-obstructive factors.

Intersecting Mechanisms of Prostatic Growth

1. Insulin and IGF Signaling

Hyperinsulinemia and elevated IGF-1 levels are central in linking metabolic disorders with increased prostatic growth [17]. Insulin and IGF-1 receptors are expressed on prostatic epithelial and stromal cells. Activation of these receptors triggers downstream signaling pathways such as phosphoinositide 3-kinase (PI3K)/Akt and mitogen-activated protein kinase (MAPK), promoting cell proliferation and survival [18]. Insulin also affects sex hormone dynamics by reducing sex hormone-binding globulin (SHBG), thereby increasing free testosterone and potentially DHT-key drivers of prostatic growth. Although the androgenic influence on BPH is well established, insulin's modulation of androgen availability adds an important layer to the metabolic hypothesis of BPH pathogenesis.

2. Chronic Inflammation

Chronic low-grade inflammation is a common denominator in MetS, diabetes, and BPH. Adipose tissue, particularly visceral fat, secretes pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α), interleukin-6 (IL-6), and monocyte chemoattractant protein-1 (MCP-1)[19]. These cytokines contribute to systemic inflammation that can affect distant organs, including the prostate. Histological analyses of BPH tissues often reveal inflammatory infiltrates consisting of T-cells and macrophages[20]. These immune cells produce growth factors and matrix metalloproteinases (MMPs) that influence stromal remodeling and epithelial proliferation. Moreover, inflammation promotes oxidative stress, further potentiating cellular damage and prostatic hyperplasia [21]. In diabetic and insulin-resistant states, inflammatory processes are amplified. Hyperglycemia upregulates nuclear factor-kappa B (NF-κB) signaling, a master regulator of inflammatory gene expression, which may accelerate prostatic tissue changes.

3. Oxidative Stress and Reactive Metabolites

Oxidative stress-an imbalance between ROS production and antioxidant defenses-is enhanced in both MetS and diabetes [22]. High glucose levels and dysregulated lipid metabolism lead to increased ROS generation via mitochondrial dysfunction and enzyme systems such as NADPH oxidases. ROS can damage cellular components, trigger pro-growth signaling pathways, and activate pro-inflammatory transcription factors [23]. In the prostate, oxidative stress has been associated with DNA damage, cellular senescence, and promotion of a pro-mitogenic environment. The presence of oxidative markers in BPH tissues supports the role of redox imbalance in disease progression.

4. Hormonal and Adipokine Dysregulation

Metabolic dysfunction alters levels of hormones and adipokines that may influence prostate biology. Leptin, an adipocyte-derived hormone, is often elevated in obesity and has pro-inflammatory and pro-mitogenic effects via

signaling through the Janus kinase (JAK)/signal transducer and activator of transcription (STAT) pathway [24]. Conversely, adiponectin, which has anti-inflammatory and insulin-sensitizing properties, is typically reduced in MetS. Sex steroid hormones, including testosterone and estrogens, are also affected by metabolic status. Aromatase activity in adipose tissue converts androgens to estrogens, potentially altering the balance of estrogen receptor-mediated effects in prostatic tissue [25]. Estrogen receptor alpha (ERα) activation has been associated with prostatic inflammation and hyperplasia in animal models. Thus, hormonal imbalances in MetS and diabetes may synergistically contribute to prostatic growth through multiple receptor-mediated pathways.

5. Autonomic Nervous System Dysfunction

The autonomic nervous system plays a role in both metabolic regulation and lower urinary tract function [26]. Insulin resistance and hyperglycemia can impair autonomic function, leading to altered sympathetic and parasympathetic activity. Enhanced sympathetic tone has been associated with prostatic smooth muscle contraction and LUTS, while diabetic autonomic neuropathy can compromise bladder sensation and contractility, worsening urinary symptoms independent of prostatic size.

Clinical Implications

Risk Stratification and Screening

Given the epidemiological links between MetS, diabetes, and BPH, clinicians should consider metabolic health when evaluating men with LUTS [27]. Screening for MetS components, glycemic control, and cardiovascular risk factors may identify patients at higher risk for rapid BPH progression. Integrating urological assessment with metabolic evaluation has the potential to improve early detection and personalized management [28].

Impact on Treatment Response

Emerging evidence suggests that metabolic dysfunction may influence response to conventional BPH therapies [29]. For example, men with insulin resistance may have reduced responsiveness to alpha-blockers or 5-alpha reductase inhibitors. Metabolic inflammation and hormonal imbalances could blunt pharmacological effects on prostatic tissues [30]. This underscores the need for integrative approaches that target underlying metabolic abnormalities alongside urological treatment.

Lifestyle and Pharmacological Interventions

Lifestyle modifications-weight reduction, increased physical activity, and dietary improvements-benefit both metabolic health and lower urinary tract symptoms [31]. Weight loss has been associated with reduced prostate volume and LUTS severity in some studies. Furthermore, medications used in diabetes management, such as metformin, exhibit anti-inflammatory and insulin-sensitizing effects that could theoretically mitigate prostatic growth, though direct clinical evidence remains limited [32]. Statins, commonly prescribed for dyslipidemia, have shown potential in reducing prostatic inflammation and symptom progression in observational studies [33]. Anti-inflammatory and antioxidant strategies represent a promising area of investigation, targeting mechanistic pathways shared by MetS, diabetes, and BPH.

Future Directions

Future directions in this field should focus on clarifying the complex biological links between metabolic dysfunction and prostatic growth to inform more targeted and effective therapies [34]. Detailed molecular profiling of prostatic tissues from men with metabolic syndrome and diabetes is needed to delineate key signaling networks, including insulin and IGF pathways, inflammatory cascades, and redox-sensitive mechanisms that drive epithelial and stromal hyperplasia [35]. Longitudinal clinical studies are essential to determine how sustained metabolic control, through lifestyle modification or pharmacological intervention, influences prostate growth dynamics, symptom progression, and response to standard BPH therapies [36]. In parallel, well-designed randomized clinical trials should evaluate whether metabolic agents such as insulin sensitizers, lipid-lowering drugs, or anti-inflammatory therapies can directly ameliorate lower urinary tract symptoms or reduce prostate volume [37]. Finally, the identification and validation of reliable metabolic and inflammatory biomarkers may enable early risk stratification and support personalized, integrated management strategies that address both metabolic health and urological outcomes.

CONCLUSION

Benign prostatic hyperplasia is a multifactorial condition that extends beyond the traditional realms of aging and androgenic stimulation. Metabolic syndrome and diabetes intersect through shared mechanisms-insulin/IGF signaling, chronic inflammation, oxidative stress, hormonal dysregulation, and autonomic dysfunction-to influence prostatic growth and symptomatology. Recognizing BPH as part of a broader metabolic-inflammatory continuum offers new perspectives on risk assessment, individualized therapy, and preventive medicine. Addressing systemic metabolic abnormalities may not only mitigate cardiovascular risk but also improve urological health in aging men.

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