

Research Progress on the Correlation between Metabolic Syndrome and Benign Prostatic Hyperplasia

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Abstract: Metabolic syndrome is a group of clinical manifestations of metabolic abnormalities, which is closely related to the occurrence of various diseases and has become an important field in modern medical research. With the change of lifestyle, the incidence of metabolic syndrome has been increasing year by year. Research shows that its components, including obesity, insulin resistance, hypertension and dys-lipidemia, may have a potential correlation with benign prostatic hyperplasia (BPH). Benign prostatic hyperplasia is a common male urinary system disease, and its occurrence mechanism is complex, involving multiple endocrine and metabolic pathways. Existing research has gradually revealed the role of the components of metabolic syndrome in the development of prostate hyperplasia, especially in aspects such as inflammatory response, changes in hormone levels and cell proliferation. However, although there have been preliminary research results, the specific mechanism between metabolic syndrome and prostate hyperplasia still needs to be further explored. Therefore, this review aims to systematically analyze the relationship between metabolic syndrome and its components and prostate hyperplasia, summarize the relevant mechanisms, and summarize the current research progress and future research directions, in order to provide a reference for clinical prevention and treatment.

Keywords: Metabolic Syndrome; Benign Prostatic Hyperplasia; Obesity; Insulin Resistance; Hypertension; Dyslipidemia

Introduction

In today's world, with the change of people's lifestyles and the acceleration of the aging process, Metabolic Syndrome (abbreviated as MS) has become a prominent health issue in the global public health field. It is characterized by a series of metabolic abnormalities such as central obesity, dyslipidemia, hypertension, and elevated blood glucose. It not only seriously affects the quality of life of individuals but is also closely related to the risk of developing various chronic diseases.

Benign Prostatic Hyperplasia (abbreviated as BPH), as a common urinary system disease in middle-aged and elderly men, has an increasing incidence year by year. Lower urinary tract symptoms, such as frequent urination, urgent urination, and dysuria, bring a lot of troubles to patients and greatly reduce their quality of life.

In recent years, more and more studies have focused on the potential associations between Metabolic Syndrome and its individual components and prostate hyperplasia. Exploring the internal connections between the two is of great significance for deeply understanding the pathogenesis of diseases, early identifying high-risk populations, and developing innovative and effective prevention and treatment strategies. On the one hand, factors encompassed by Metabolic Syndrome, such as obesity, hyperlipidemia, hypertension, and hyperglycemia, may affect the microenvironment of prostate tissue, the balance of cell proliferation and apoptosis through complex endocrine, inflammatory, and metabolic pathway disorders, and thus promote the occurrence and development of prostate hyperplasia. On the other hand, whether the local pathophysiological changes related to prostate hyperplasia will, in turn, act on the body's metabolic state and exacerbate the process of Metabolic Syndrome is also worthy of in-depth analysis. This review aims to comprehensively sort out the cutting-edge research achievements in this field in recent years, so as to provide a solid theoretical basis for further subsequent research and clinical practice and contribute to new breakthroughs in related fields.

1. The Definition and Diagnostic Criteria of Metabolic Syndrome

In 1998, Alberti et al. first established the definition of metabolic syndrome and proposed that insulin resistance was an absolute requirement for diagnosing metabolic syndrome, along with meeting two additional criteria, which specifically covered obesity, dyslipidemia, hypertension, and microalbuminuria ^[1]. Since then, various associations and organizations have provided different definitions for diagnosing metabolic syndrome. In 2005, Grundy et al. updated the definition of metabolic syndrome in the National Cholesterol Education Program (NCEP) Adult Treatment Panel III (ATP III), which has become one of the most widely used diagnostic criteria. According to the NCEP ATP III definition, a diagnosis of metabolic syndrome can be made if three or more of the following five indicators are met: a waist circumference greater than 40 inches (for men) or 35 inches (for women), blood pressure exceeding 130/85 mmHg, fasting triglyceride (TG) levels above 150 mg/dl, fasting high-density lipoprotein (HDL) cholesterol levels below 40 mg/dl (for men) or 50 mg/dl (for women), and fasting blood glucose above 100 mg/dl ^[2].

2. Correlation between components of metabolic syndrome and benign prostatic hyperplasia

2.1 Obesity and benign prostatic hyperplasia

Obesity plays an important role in the occurrence and development of benign prostatic hyperplasia. Many studies have shown that there is a significant positive correlation between obesity and benign prostatic hyperplasia. Specifically, obesity indicators such as body mass index (BMI) and waist circumference have been shown to be associated with prostate volume, the occurrence of clinical BPH, and the severity of lower urinary tract symptoms. A recent Mendelian randomization study showed a link between body mass index (BMI) and prostate volume ^[3]. In another prospective cohort study, Negi et al analyzed the data of 560 BPH patients, and the results also suggested that there was a significant correlation between BMI and obesity ^[4]. The control study results of Guorui et al showed that obese BPH patients have unique metabolic characteristics. 17 kinds of poor metabolism foreign bodies were detected in obese and non obese BHP patients, among which the changes of phosphatidylethanolamine (PE) and phosphatidylcholine (PC) are related to the occurrence and progression of BPH ^[5]. Scholars in Taiwan have proposed that waist circumference \geq 90cm is an independent risk factor for benign prostatic hyperplasia. Men with abdominal obesity have twice the risk of benign prostatic volume when excluding the influence of significant obesity related metabolic diseases. The prostate volume of men with BMI \geq 25 kg/m2 was significantly larger than that of men with BMI <23 kg/m2, and the prostate volume of men with waist circumference >90 cm was significantly larger than that of men with waist circumference \leq 90 cm. Waist circumference >90 cm is an independent risk factor for benign circumference \leq 90 cm is an independent risk factor for benign circumference \leq 90 cm. Waist circumference >90 cm is an independent risk factor for benign prostate volume of men with waist circumference \leq 90 cm was significantly larger than that of men with waist circumference \leq 90 cm. Waist circumference >90 cm is an independent ris

2.2 Elevated blood glucose, insulin resistance and prostatic hyperplasia

Elevated blood glucose and insulin resistance are also closely related to the occurrence of benign prostatic hyperplasia. Zhou et al analyzed the correlation between glucose metabolism, insulin resistance and inflammatory factors and IPSS by logistic regression, and concluded that fasting blood glucose and fasting insulin were closely related to IPSS score, which could reflect the severity and prognosis of BPH ^[8]. In Ribeiro's study, it was found that obesity can cause structural and ultrastructural changes in rat prostate epithelium; These changes may affect gland homeostasis and physiological function. The epithelial and smooth muscle cell hyperplasia and increased FGF-2 expression observed in this experimental model of obesity / insulin resistance may explain the high frequency of benign prostatic hyperplasia in insulin resistant men ^[9]. In the prospective study of ozcan et al., it was found that the prostate volume of diabetic patients was larger. And concluded that the large prostate volume of patients with benign prostatic hyperplasia was positively correlated with the diagnosis of diabetes ^[10]. In another cross-sectional study, abnormal glucose metabolism was found as a decisive factor to drive prostate volume enlargement ^[11]. In a recent meta-analysis, researchers included 1685 BPH patients with diabetes and 4653 BPH patients without diabetes for analysis and research. The results showed that BPH patients with diabetes had significantly higher lower urinary tract symptoms than BPH patients without diabetes ^[12].

2.3 Hypertension and benign prostatic hyperplasia

Hypertension and benign prostatic hyperplasia (BPH) are both common and age-related diseases in men. Although hypertension is one of the important diagnostic criteria of metabolic syndrome (MS), its relationship with BPH is controversial, but the incidence of both increas-

es with age. Clinical epidemiological survey shows that elderly patients with BPH are often complicated with hypertension, and hypertension is an independent risk factor for the occurrence and progression of BPH. An observational study showed that a history of BPH was associated with a higher risk of cardiovascular disease (CVD)^[13]. In another prospective study, after 3 years of follow-up, Fu et al found that hypertension may accelerate the clinical progression of BPH ^[14]. Sugaya et al found in their study that hypertension may worsen lower urinary tract symptoms and may reduce the improvement of BPH symptoms by drugs ^[15]. In a single center cross-sectional study, G ü ven et al used Pearson correlation and linear regression analysis to find that systolic blood pressure was positively correlated with all urinary storage symptoms ^[16]. However, in a recent study, researchers compared the clinical and physiological parameters of BPH in hypertensive and normotensive patients and found that there may be no significant association between hypertension and prostate volume ^[17].

2.4 Dyslipidemia and prostatic hyperplasia

Dyslipidemia refers to the increase or decrease of serum cholesterol (TG), triglyceride (TC), low-density lipoprotein (LDL-C), high-density lipoprotein (HDL-C). Dyslipidemia is also an important part of metabolic syndrome, which is closely related to the occurrence and development of BPH. The results of Zhu et al showed that tg/hdl-c and tc/hdl-c were associated with BPH risk. Tg/hdl-c was a strong independent risk factor for BPH in Chinese adults. Male subjects with normal tg/hdl-c levels should pay attention to higher tg/hdl-c ratio ^[18]. The study of Shih et al similarly showed that hyperlipidemia was associated with an increased risk of clinical BPH ^{^[19]. Shen et al found in their study that both high LDL and low HDL were important predictors of BPH severity ^{^[20]. Some small-scale clinical trials tried to use lipid-lowering drugs to treat patients with benign prostatic hyperplasia complicated with dyslipidemia. It was observed in some cases that with the improvement of blood lipid indicators, patients' lower urinary tract symptoms were relieved, and the growth rate of prostate volume was also slowed down ^[21].}}

Summary

The correlation between metabolic syndrome and benign prostatic hyperplasia revealed the key role of metabolic abnormalities in the pathogenesis of benign prostatic hyperplasia. Recent studies have shown that components of metabolic syndrome, such as obesity, insulin resistance and dyslipidemia, may affect the physiological function of the prostate through a variety of mechanisms, thereby promoting the occurrence of benign prostatic hyperplasia. This finding not only provides us with a new perspective on the etiology of benign prostatic hyperplasia, but also provides potential prevention and treatment directions for clinic. In the current study, there are some differences in the views and findings of different scholars. For example, some studies pointed out that the severity of metabolic syndrome was positively correlated with the risk of benign prostatic hyperplasia, while others emphasized that the specific components of metabolic abnormalities had a more significant impact on benign prostatic hyperplasia. This difference may originate from many factors such as study design, sample selection and regional differences. Therefore, future research needs to be carried out in larger groups to verify and integrate different research results, so as to provide a more comprehensive understanding. In addition, in-depth analysis of the mechanism between metabolic syndrome and benign prostatic hyperplasia will be an important direction of future research. This will not only help reveal potential biomarkers, but also provide a theoretical basis for clinical development of personalized intervention strategies. For example, early intervention for metabolic abnormalities may help to reduce the incidence of benign prostatic hyperplasia and bring a positive impact on the quality of life of patients. In summary, the relationship between metabolic syndrome and benign prostatic hyperplasia provides a new perspective for us to understand the complexity of prostate disease. Through further research, we hope to find a better balance between metabolic management and prostate health, and open up a new way for clinical practice.

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