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Article

Relationship Between Prostate-Specific Antigen Levels And Metabolic Syndrome In Patients With Benign Prostatic Hyperplasia And Prostate Cancer

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Abstract: Prostate-specific antigen (PSA) is the most widely used biomarker for the diagnosis, risk assessment, and clinical monitoring of benign prostatic hyperplasia (BPH) and prostate cancer (PCa). Nevertheless, serum PSA concentrations are influenced not only by pathological changes in the prostate gland but also by systemic metabolic abnormalities. Metabolic syndrome, characterized by central obesity, impaired glucose metabolism, dyslipidemia, and arterial hypertension, has been increasingly recognized as an important modifier of prostate physiology through its effects on chronic inflammation, insulin resistance, oxidative stress, and hormonal imbalance. These metabolic disturbances may alter PSA secretion and reduce the diagnostic accuracy of PSA-based screening, thereby complicating the early detection and clinical evaluation of prostate diseases. However, the relationship between serum PSA levels and metabolic syndrome in patients with BPH and PCa remains incompletely understood. The present study aimed to evaluate the association between serum prostate-specific antigen levels and metabolic syndrome in patients with benign prostatic hyperplasia and prostate cancer by analyzing clinical, anthropometric, and biochemical characteristics. The study included patients diagnosed with BPH, patients with histologically confirmed prostate cancer, and an age-matched healthy control group. Clinical evaluation comprised body mass index, waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, serum triglycerides, high-density lipoprotein cholesterol, prostate volume measured by transrectal ultrasonography, and serum PSA concentration. Comparative statistical analyses were performed to determine the relationship between metabolic syndrome components and PSA levels in different study groups.

Keywords: prostate-specific antigen, metabolic syndrome, benign prostatic hyperplasia, prostate cancer, obesity, dyslipidemia, insulin resistance, prostate volume, biomarkers, risk assessment.

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1. Introduction

Benign prostatic hyperplasia (BPH) and prostate cancer (PCa) are among the most common urological diseases affecting aging men and represent a substantial global public health burden. Benign prostatic hyperplasia is the leading cause of lower urinary tract symptoms in elderly males, whereas prostate cancer remains one of the most frequently diagnosed malignancies and a major cause of cancer-related mortality worldwide. According to recent epidemiological reports, the incidence of both conditions continues to increase owing to population aging, prolonged life expectancy, and improvements in diagnostic techniques. Although BPH and PCa are distinct pathological entities, they share several common risk factors, including advancing age, hormonal imbalance, chronic inflammation, genetic susceptibility, and metabolic disturbances[1]. Prostate-specific antigen (PSA) is a glycoprotein enzyme produced primarily by the epithelial cells of the prostate gland and remains the most widely used biomarker for the diagnosis, risk assessment, and clinical monitoring of prostate diseases. Since its introduction into clinical practice, serum PSA measurement has significantly improved the early detection of prostate cancer and has become an essential component of screening programs, treatment monitoring, and follow-up evaluation.

Elevated PSA concentrations are commonly associated with prostate cancer; however, increased PSA levels may also occur in patients with benign prostatic hyperplasia, prostatitis, urinary tract infections, and following various urological procedures. Consequently, interpretation of PSA values requires careful consideration of multiple clinical and biological factors that may influence its serum concentration[2].

In recent years, increasing attention has been directed toward the influence of metabolic syndrome on prostate physiology and PSA levels. Metabolic syndrome is a complex metabolic disorder characterized by the coexistence of central obesity, insulin resistance, impaired glucose metabolism, dyslipidemia, and arterial hypertension. These abnormalities are associated with chronic low-grade inflammation, oxidative stress, endothelial dysfunction, and hormonal imbalance, all of which may contribute to structural and functional alterations of the prostate gland. The global prevalence of metabolic syndrome has risen markedly during the past decades, making it one of the most important public health challenges and an increasingly recognized modifier of prostate disease development and progression[3].

Several biological mechanisms have been proposed to explain the relationship between metabolic syndrome and alterations in PSA levels. Excess visceral adipose tissue secretes numerous pro-inflammatory cytokines, including tumor necrosis factor-α, interleukin-6, and leptin, while reducing the production of adiponectin, an adipokine with anti-inflammatory and antiproliferative properties. These changes promote chronic systemic inflammation, oxidative stress, and activation of intracellular signaling pathways involved in cellular proliferation and apoptosis. Furthermore, insulin resistance and compensatory hyperinsulinemia increase circulating insulin-like growth factor-1 (IGF-1), which stimulates prostate epithelial cell proliferation and may facilitate both benign hyperplastic growth and malignant transformation. Metabolic abnormalities may also affect androgen metabolism, vascular function, and prostate tissue remodeling, thereby influencing PSA synthesis and secretion[4].

Dyslipidemia and impaired glucose metabolism constitute additional mechanisms through which metabolic syndrome may modify PSA concentrations. Elevated serum triglycerides, reduced high-density lipoprotein cholesterol, and chronic hyperglycemia contribute to oxidative damage, mitochondrial dysfunction, and persistent inflammatory activation within prostate tissue. Obesity-related hormonal alterations, including decreased testosterone concentrations and increased aromatization of androgens to estrogens, may further influence prostate growth and PSA production. Consequently, patients with metabolic syndrome may exhibit PSA levels that do not accurately reflect the underlying pathological status of the prostate, potentially affecting the sensitivity and specificity of PSA-based diagnostic strategies[5].

Although numerous studies have investigated the relationship between individual components of metabolic syndrome and prostate diseases, their findings remain inconsistent. Some investigators have reported higher PSA concentrations among men with metabolic syndrome, whereas others have observed lower or unchanged PSA levels, particularly in obese individuals. These discrepancies may be explained by differences in study design, ethnicity, patient characteristics, diagnostic criteria, and the complex interactions between metabolic, hormonal, and inflammatory pathways. Moreover, relatively few studies have simultaneously compared the relationship between PSA levels and metabolic syndrome in patients with benign prostatic hyperplasia and prostate cancer, limiting the understanding of the clinical significance of metabolic abnormalities across different prostate diseases[6].

A better understanding of the interaction between PSA and metabolic syndrome is of considerable clinical importance. Recognition of metabolic factors that influence PSA concentrations may improve the interpretation of laboratory findings, reduce diagnostic inaccuracies, enhance risk stratification, and contribute to more individualized clinical

decision-making. Furthermore, identifying metabolic determinants of PSA variability may help optimize prostate cancer screening strategies and improve the management of patients with benign prostatic hyperplasia and prostate cancer[7].

Therefore, the present study aimed to evaluate the relationship between serum prostate-specific antigen levels and metabolic syndrome in patients with benign prostatic hyperplasia and prostate cancer by comprehensively analyzing anthropometric, biochemical, and clinical characteristics. The findings of this study may provide additional evidence regarding the influence of metabolic syndrome on PSA levels and contribute to the development of more accurate diagnostic and personalized management strategies for patients with prostate diseases[8].

2. Materials And Methods

This cross-sectional clinical study was conducted at the multidisciplinary clinic of Andijan State Medical Institute and included a total of 436 male participants. The study population consisted of 217 patients diagnosed with prostate diseases, including benign prostatic hyperplasia (BPH) and prostate cancer (PCa), while 219 age-matched apparently healthy men without clinical or instrumental evidence of prostatic pathology served as the control group. The diagnosis of benign prostatic hyperplasia was established according to the recommendations of the European Association of Urology based on lower urinary tract symptoms, digital rectal examination, prostate-specific antigen (PSA), transrectal ultrasonography, and uroflowmetry. Prostate cancer was diagnosed according to current European Association of Urology guidelines using digital rectal examination, serum PSA concentration, transrectal ultrasonography, multiparametric magnetic resonance imaging when indicated, and histopathological confirmation following transrectal ultrasound-guided prostate biopsy[9].

Metabolic syndrome was diagnosed according to the International Diabetes Federation (IDF) criteria. Central obesity was considered the principal diagnostic criterion and was accompanied by at least two additional metabolic abnormalities, including elevated fasting plasma glucose, hypertriglyceridemia, reduced high-density lipoprotein cholesterol (HDL-C), and arterial hypertension. According to the presence or absence of metabolic syndrome, patients with benign prostatic hyperplasia and prostate cancer were further classified into subgroups for comparative analysis[10].

Comprehensive clinical assessment included age, body mass index (BMI), waist circumference, systolic and diastolic blood pressure, fasting plasma glucose, serum triglycerides, total cholesterol, low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), serum prostate-specific antigen (PSA), and prostate volume determined by transrectal ultrasonography. Anthropometric measurements were performed using standardized techniques. Body mass index was calculated as body weight divided by height squared (kg/m²), while waist circumference was measured at the midpoint between the lower rib margin and the iliac crest. Blood pressure was measured twice after at least 10 minutes of rest, and the average value was used for statistical analysis. Prostate volume was calculated using the standard ellipsoid formula based on transrectal ultrasonographic measurements[11].

Venous blood samples were collected from all participants after an overnight fast of 8–12 hours. Serum concentrations of fasting plasma glucose, total cholesterol, triglycerides, HDL cholesterol, LDL cholesterol, and PSA were determined using automated biochemical analyzers and commercially available reagent kits according to the manufacturers' instructions. Serum PSA concentrations were measured by a chemiluminescent immunoassay in the certified clinical laboratory of the institution. Internal quality-control procedures were implemented throughout the study to ensure analytical accuracy and reproducibility.

The primary outcome of the study was to evaluate the relationship between serum PSA concentration and metabolic syndrome in patients with benign prostatic hyperplasia and

prostate cancer. Associations between PSA levels and individual metabolic syndrome components, including obesity, fasting plasma glucose, serum triglycerides, HDL cholesterol, arterial hypertension, and prostate volume, were assessed to determine the potential influence of metabolic abnormalities on PSA concentration and its clinical interpretation[12].

Statistical analyses were performed using IBM SPSS Statistics software (version 26.0). Continuous variables were expressed as mean ± standard deviation (SD), whereas categorical variables were presented as frequencies and percentages. The Shapiro–Wilk test was applied to assess normality of data distribution. Differences between study groups were analyzed using the independent-samples t-test or one-way analysis of variance (ANOVA) for normally distributed variables and the Mann–Whitney U test or Kruskal–Wallis test for non-normally distributed variables. Pearson's chi-square (χ^2) test was used to compare categorical variables. Correlations between serum PSA levels and metabolic parameters were evaluated using Pearson's or Spearman's correlation coefficients, as appropriate. Multivariate linear regression analysis was performed to identify independent predictors of serum PSA concentration after adjustment for potential confounding variables. A two-sided p value of less than 0.05 was considered statistically significant.

3. Results

A total of 436 men were included in the present study, comprising patients with benign prostatic hyperplasia (BPH), prostate cancer (PCa), and an age-matched healthy control group. Comparative analysis demonstrated that metabolic syndrome was significantly more prevalent among patients with prostate diseases than among healthy individuals. The prevalence of metabolic syndrome was highest in patients with prostate cancer, followed by those with benign prostatic hyperplasia, indicating a close association between metabolic disturbances and prostate pathology[13].

Anthropometric assessment revealed significant differences between study groups. Patients with metabolic syndrome exhibited significantly higher body mass index (BMI) and waist circumference than participants without metabolic syndrome ($p<0.05$). Similarly, systolic and diastolic blood pressure values were significantly elevated in patients with metabolic syndrome, confirming the progressive deterioration of cardiovascular and metabolic status in these individuals[14].

Biochemical evaluation demonstrated marked alterations in glucose and lipid metabolism among patients with metabolic syndrome. Fasting plasma glucose and serum triglyceride concentrations were significantly higher, whereas high-density lipoprotein cholesterol (HDL-C) levels were significantly lower in patients with metabolic syndrome compared with metabolically healthy participants ($p<0.05$). Total cholesterol and low-density lipoprotein cholesterol (LDL-C) also tended to be higher in patients with metabolic abnormalities, reflecting the presence of dyslipidemia associated with metabolic syndrome[15].

Serum prostate-specific antigen (PSA) concentrations differed significantly according to metabolic status. Patients with metabolic syndrome demonstrated higher PSA levels than patients without metabolic syndrome, particularly in the prostate cancer group. Likewise, prostate volume measured by transrectal ultrasonography was significantly greater among patients with metabolic syndrome than among metabolically healthy individuals. These findings indicate that metabolic abnormalities are associated with increased PSA concentrations and enlargement of the prostate gland.

Further analysis of individual components of metabolic syndrome demonstrated positive associations between serum PSA concentration and several metabolic parameters. PSA levels increased progressively with increasing body mass index, waist circumference, fasting plasma glucose, serum triglycerides, and prostate volume. In contrast, serum HDL cholesterol showed an inverse relationship with PSA concentration, indicating that

lower HDL-C levels were associated with higher PSA values[16].

Comparative analysis between patients with benign prostatic hyperplasia and prostate cancer demonstrated that serum PSA concentrations were significantly higher in the prostate cancer group regardless of metabolic status. However, patients with prostate cancer who also had metabolic syndrome exhibited the highest PSA concentrations among all study groups. Similarly, prostate volume was significantly greater in patients with metabolic syndrome than in those without metabolic abnormalities, particularly among individuals with benign prostatic hyperplasia[17].

Correlation analysis demonstrated significant positive relationships between serum PSA concentration and body mass index, waist circumference, fasting plasma glucose, serum triglycerides, and prostate volume ($p<0.05$). Conversely, HDL cholesterol demonstrated a significant negative correlation with serum PSA levels. These findings indicate that progressive metabolic dysfunction is accompanied by increasing PSA concentrations and more pronounced clinical manifestations of prostate disease[18].

Multivariate linear regression analysis identified several independent determinants of serum PSA concentration. Age, body mass index, waist circumference, fasting plasma glucose, serum triglycerides, prostate volume, and the presence of metabolic syndrome remained independently associated with elevated PSA levels after adjustment for potential confounding variables. Among these variables, prostate volume and metabolic syndrome demonstrated the strongest independent associations with serum PSA concentration[19-20].

4. Conclusion

The present study demonstrates that metabolic syndrome is closely associated with increased serum PSA concentrations and larger prostate volume in patients with benign prostatic hyperplasia and prostate cancer. These findings suggest that metabolic abnormalities should be considered when interpreting serum PSA levels, as they may influence the diagnostic performance of PSA and contribute to more accurate clinical assessment and individualized management of patients with prostate diseases.

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