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Investigation of Galectin-3 and Glucagon-Like Peptide (9-36) Amide Levels in Patients Diagnosed with Benign Prostatic Hyperplasia

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Abstract

Introduction: Prostate-specific antigen (PSA) is a standard laboratory assay employed in the diagnosis of benign prostatic hyperplasia (BPH). Nonetheless, alternative diagnostic biomarkers are under investigation. This study aimed to examine the possibility of blood levels of galectin-3 (Gal-3) and glucagon-like peptide-1 (GLP-1) (9-36) amide as alternative diagnostic indicators for BPH.

Materials and Methods: A control group comprising 42 healthy individuals without complaints and 42 individuals diagnosed with BPH, aged over 40, exhibiting prostate-related lower urinary tract symptoms for a minimum of 6 months, an IPSS score exceeding 13, a peak urine flow rate of 4-15 mL/s, and a total PSA level below 3 ng/mL, was incorporated into the study as the patient cohort.

Results: Gal-3 levels were markedly higher in the patient group than in the control group (median values 7.6 ng/dL vs. 5.17 ng/dL; $p = 0.015$). Nonetheless, GLP-1 levels did not differ significantly between the two groups (median values 15.96 pmol/L vs. 13.58 pmol/L; $p = 0.424$). Free PSA levels (f-PSA) and the f-PSA-to-total PSA (t-PSA) ratio (f-PSA/t-PSA) were significantly increased in the BPH group compared to the controls ($p = 0.0001$ and $p = 0.002$, respectively), while t-PSA levels did not differ significantly between the groups ($p = 0.092$).

Conclusion: In summary, increased Gal-3 levels in BPH may significantly contribute to disease pathogenesis. This study is significant as it represents the first investigation into the importance of both parameters in patients diagnosed with benign prostatic hyperplasia.

Key words: Benign prostatic hyperplasia; galectin-3; glucagon-like peptide-1 (9-36) amide; prostate-specific antigen.

Introduction

Benign Prostatic Hyperplasia (BPH) represents the most prevalent urological condition among men and is defined histopathologically as a non-malignant hyperplastic process involving epithelial and stromal components, leading to progressive prostate enlargement. In its early phase, hyperplastic nodules originate in the periurethral region and proliferate incrementally. The subsequent phase, predominantly affecting men over 60 years of age, is marked by a rapid and concurrent expansion of glandular nodules. Although many factors have been implicated to date, the true cause of BPH is not precisely known and is considered a multifactorial event. Galectin-3 (Gal-3) is widely distributed and found in multiple intracellular and extracellular locations, and it has important biological and immune functions. Importantly, prostate-specific antigen (PSA) is capable of cleaving to Gal-3. Despite this interaction, current evidence suggests that PSA and Gal-3 function independently within the molecular pathways underlying prostate carcinogenesis. Gal-3 has been implicated in several key mechanisms associated with tumor development and progression. In the context of prostate cancer, Gal-3's contribution to tumor cell survival, immune evasion, and metastatic spread positions it as a potential biomarker and therapeutic target. Glucagon-like peptide-1 (GLP-1) functions as an incretin hormone that has a low molecular weight and a very short half-life. It is produced and released by enteroendocrine L cells. The prohormone convertase enzyme yields two biologically active isoforms: GLP-1 (7–36) amide, which constitutes the predominant circulating form, and GLP-1 (7–37), a less abundant but functionally relevant variant. The inactive degradation products of GLP-1 by dipeptidyl peptidase-4 (DPP-4) are GLP-1 (9–36) amide and GLP-1 (9–37). These metabolites have minimal receptor affinity and are subsequently eliminated through renal clearance mechanisms. GLP-1

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mediates its physiological functions by binding to GLP-1 receptors (GLP1-R). Our review of the literature revealed no existing studies examining the association between serum Gal-3 and GLP-1 levels in benign prostatic hyperplasia (BPH). This study aimed to assess the differences in serum Gal-3 and GLP-1 concentrations among individuals with PSA levels < 3 ng/mL who were clinically diagnosed with BPH by a specialist.

Materials and Methods

Study design and patients: This study was reviewed and approved by the Institutional Review Board at the University of Ordu and was conducted under the principles of the Declaration of Helsinki. Ethics committee approval was obtained from the Ordu University Local Ethical Committee under Reference No: 2022/278. The study is cross-sectional and enrolled 84 patients who visited the urology outpatient clinic between January 2023 and January 2024. The study population consisted of two cohorts: a control group comprising 42 asymptomatic, healthy volunteers and a patient group of 42 individuals clinically diagnosed with benign prostatic hyperplasia (BPH) by a specialist. Individuals who declined participation or withdrew from the study, those diagnosed with diabetes, hypertension, or acute infections and receiving treatment for these conditions, patients with a history of urethral stricture, urological surgery, pelvic radiotherapy, or urological malignancies, as well as those using GLP-1 receptor agonists or antagonists, were excluded from the study. Eligible participants consisted of men over 40 who had experienced prostate-related lower urinary tract symptoms for a minimum of six months, with an International Prostate Symptom Score (IPSS) exceeding 13, a peak urine flow rate ranging from 4 to 15 mL/s, and a total PSA level below 3.0 ng/mL. Blood samples were taken after fasting for at least 8 hours into anticoagulant free tube (BD Vacutainer SST-II Advance, UK) and permitted to coagulate at ambient temperature for 30 minutes. Thereafter undergoing centrifugation at 2000 × g for 10 minutes (Nuve 1200R, Türkiye). The serum was divided into Eppendorf tubes (Ayset, Türkiye) and preserved at –80°C until analysis.

Biochemical analysis: The values of PSA, biochemistry, and hormone parameters included in our study were obtained from the patients file information by following the test results requested simultaneously with the blood samples taken for the parameters measured in this study for the routine follow-up of the patients. The biochemistry and hormone test parameter measurements in our study were performed using commercial kits produced by Roche Diagnostics and the Hitachi Cobas c 501 and Hitachi Cobas e 801 (Roche Diagnostics Ltd., Japan) autoanalyzers. The low-density lipoprotein cholesterol (LDL-C) level was calculated using Friedewald's formula (LDL-C = total cholesterol - high-density lipoprotein cholesterol (HDL-C) - triglycerides/5). Gal-3 and GLP-1 levels were measured by the ELISA (Enzyme Linked Immunosorbent Assay) method using commercial ELISA kits (SunRedbio Co. Ltd., Shanghai/China, 201-12-7673, respectively; REF: DZE201121952 LOT: 202312 and REF: DZE201120023 LOT: 202401). The BioTek ELx800 Microplate Reader and Biotek ELx50 washer were used for the ELISA test parameter measurements (BioTek Instruments, Inc., USA).

Statistical analysis: Statistical analyses were conducted using MedCalc software. The sample size was determined by G*Power analysis, with an alpha level of 0.05 and a statistical power of 0.95. Descriptive statistics included counts, means, standard deviations, frequencies, percentages, medians, and interquartile ranges (25th and 75th percentiles). The Kolmogorov-Smirnov test was utilized to evaluate the normality of numerical data distributions. For variables adhering to a normal distribution, independent t-tests were used for group comparisons, whereas the Mann-Whitney U test was applied for data that did not conform to normality. Categorical variables were examined using the chi-square test. Relationships among variables were analyzed using Spearman's correlation analysis. A p-value below 0.05 was considered statistically significant.

Table 1: Demographic characteristics of the groups included in the study

	Control		BPH		
Parameters	**Mean**	**SD**	**Mean**	**SD**	**p**
Age (Year)	56.11	8.08	61.92	8.58	0.002 *
BMI (kg/m²)	27.05	3.38	26.85	4.12	0.837

***** Significant difference at <0.05 level with independent t-test. **Abbreviations: BMI;** body mass index, **BPH;** benign prostatic hyperplasia

Results

Table 1 summarizes the demographic characteristics of the participants. As seen in this

table, BPH patients were statistically older than controls (p=0.002). Comparisons of Gal-3, GLP-1, free PSA (f-PSA), and total PSA (t-PSA) values of both groups are presented in Table 2 and Figures 1-3. Gal-3 (p=0.015), f-PSA (p=0.0001), and f-/t-PSA ratio (p=0.002) were significantly higher in the BPH group, but GLP-1 (p=0.424) and t-PSA (p=0.092) values did not show any significant differences between the groups.

Table 2: Comparison of Gal-3, GLP-1, free PSA, total PSA and free/total PSA levels of groups.

	Control					BPH					
	Mean	**SD**	**Median**	**25P**	**75P**	**Mean**	**SD**	**Median**	**25P**	**75P**	p
Gal-3 (ng/mL)	8.04	7.17	5.17	4.1	8.6	9.67	5.79	7.6	6.0	12.0	0.015+
GLP-1 (pmol/L)	20.69	19.69	13.58	10.7	22	25.22	20.62	15.961	11	34.4	0.424
t-PSA (ng/mL)	1.04	0.76	0.76	0.5	1.4	1.32	0.81	1.2	0.6	1.8	0.092
f-PSA (ng/mL)	0.25	0.15	0.20	0.2	0.3	0.46	0.23	0.4	0.3	0.7	0.0001+
f-PSA/t-PSA	0.30	0.18	0.27	0.19	0.34	0.43	0.28	0.334	0.3	0.5	0.002+

+ Significant difference at <0.05 level with Mann-Whitney U Test. **Abbreviations:** Gal-3; galectin-3, GLP-1; Glucagon-like peptide-1, t-PSA; total prostate-specific antigen, f-PSA; free prostate-specific antigen, BPH; benign prostatic hyperplasia

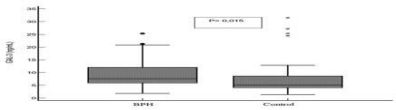

Figure 1: The average galectin-3 values of the groups.

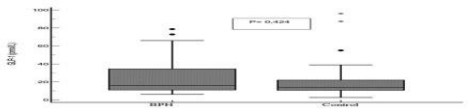

Figure 2: The average glucagon-like peptide-1 values of the groups.

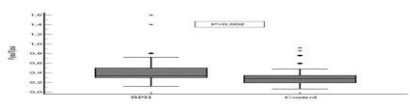

Figure 3: The average free/total prostate-specific antigen values of the groups

Table 3: Comparison of biochemical parameters of the groups.

	Control					BPH					
	Mean	**SD**	**Median**	**25P**	**75P**	**Mean**	**SD**	**Median**	**25P**	**75P**	**p**
Glucose(mg/dL)	98.00	12.65	101	89.3	105	105.7	20.16	100.9	93.8	113	0.22
BUN (mg/dL)	18.51	10.01	14.3	12.6	21.1	17.43	8.445	14.4	12.9	18.7	0.99
Creatinine (mg/dL)	1.04	1.24	0.875	0.77	0.97	1.60	4.443	0.925	0.82	1.0	0.09
e-GFR (CKD-EPI)	96.11	15.67	93.795	85.9	100.7	82.08	22.05	83.19	72.9	96.9	0.002+
CRP (mg/dL)	4.61	9.41	1.56	0.64	3.0	2.69	3.396	1.62	0.76	3.23	0.80
Uric acid (mg/dL)	5.03	1.16	5.1	4.5	5.6	5.29	0.972	5.2	4.6	6.0	0.38
Total Bilirubin (mg/dL)	0.63	0.42	0.485	0.41	0.73	0.69	0.402	0.62	0.38	0.93	0.40
Direct Bilirubin (mg/dL)	0.29	0.31	0.22	0.17	0.28	0.24	0.111	0.21	0.17	0.31	0.98
GGT (U/L	19.22	7.61	18.5	13.0	23.0	21.5	9.405	20.0	16.0	25.0	0.31
ALT (U/L)	20.99	9.240	19.3	14.0	28.0	25.76	8.71	16.95	13.0	27.0	0.48
AST (U/L)	22.66	12.70	20	15.0	25.0	24.03	14.66	19.85	17.0	25.0	0.97
ALP (U/L)	77.09	20.64	80.5	61.0	91.0	80.74	37.33	72.5	61.0	85.0	0.48
LDH (U/L	186.19	49.25	177	162.0	206	188.1	65.57	182	1470	1950	0.95
Total protein (g/L)	72.04	3.762	71.95	69.0	74.2	68.64	6.12	69.35	65.4	73	0.010+
Albumin (g/L)	46.08	7.55	46.75	45.1	48.1	45	2.96	44.1	42.6	47.4	0.017+
Magnesium (mg/dL)	2.08	0.38	2.015	1.97	2.09	2.026	0.471	1.97	1.91	2.06	0.038+
Ferritin (ng/mL)	103.25	61.44	85.05	64.5	148	104.8	69.3	95.8	48.7	140	0.91
Iron (µg/dL)	85.30	24.82	80	72.5	95.3	90.05	31.75	85.55	74.5	97.7	0.71
Folate (ng/mL)	7.33	3.01	6.285	5.55	7.7	7.07	2.164	6.955	5.74	8.51	0.43
VitaminB12 (pg/mL)	306.38	122.21	268	235	349.0	303.3	115.1	282	233	371	0.92
Calcium (mg/dL)	9.516	0.36	9.5	9.22	9.7	9.464	0.42	9.45	9.1	9.8	0.54
Cholesterol (mg/dL)	202.44	37.35	203.0	181.0	224.0	200.9	42.84	196.0	165.0	230.0	0.86
Triglyceride (mg/dL)	134.2	54.17	122.5	96.0	160.0	156.6	79.42	150.5	102.0	190.0	0.13
HDL-C (mg/dL)	48.46	14.50	43.4	38.8	55.6	45.03	10.69	42.05	38.4	53	0.33
LDL-C (mg/dL)	127.98	35.14	124.75	96.8	156	124.7	36.85	122.05	97.5	152	0.67

+ Significant difference at <0.05 level with Mann-Whitney U Test. **Abbreviations:** BUN; blood urea nitrogen, e-GFR; estimated glomerular filtration rate, CRP; C-reactive protein, GGT; gamma-glutamyl transferase, ALT; alanine amino transferase, AST; aspartate amino transferase, ALP, Alkaline phosphatase, LDH; lactate dehydrogenase, HDL-C; high-density lipoprotein-cholesterol**,** LDL-C; low-density lipoprotein-cholesterol

Comparisons of biochemical parameters are presented in Table 3. The results of the correlation analysis are shown in Table 4, and a significant positive correlation was also found between GLP-1 and Gal-3 (r=0.30, p=0.04).

Discussion

The etiology of BPH remains incompletely understood, with multiple factors believed to contribute to its development either independently or synergistically. Among the established risk

Table 4: Correlation analysis of BPH and control groups parameters.

	t-PSA	f-PSA	f-t-PSA	Gal-3	GLP-1	eGFR	Total protein	Albumin	Mg
Age	0.30*	0.36*	0.01	-0.01	-0.05	-0.46*	-0.19*	-0.30*	-0.08
t-PSA		0.64*	-0.48*	0.13	-0.08	-0.16	-0.13	-0.25*	-0.16
f-PSA			0.28*	0.17	-0.02	-0.34*	-0.16	-0.29*	-0.27*
f-PSA/t-PSA				0.09	0.07	-0.14	-0.01	-0.01	-0.18
Gal-3					0.30*	0.09	-0.14	0.16	0.03
GLP-1						0.19	-0.08	0.06	-0.01
eGFR							0.19	0.32*	0.19
Total protein								0.35*	0.04
Albumin									0.1

*: $p<0.05$ **Abbreviations:** Gal-3; galectin-3, GLP-1; glucagon-like peptide-1, t-PSA; total prostate-specific antigen, f-PSA; free prostate-specific antigen, e-GFR; estimated glomerular filtration rate, Mg; magnesium.

factors, the presence of functional tests and advancing age have been consistently implicated in BPH pathogenesis. Research also suggests that hormonal changes, particularly involving testosterone and dihydrotestosterone, play a significant role in the enlargement of the prostate. Additionally, lifestyle factors such as obesity and diet may further influence the risk and severity of BPH. Additional contributors include stromal-epithelial interactions, hormonal influences, diabetes mellitus (DM), smoking, lifestyle factors, and genetic predisposition (11, 12). Although PSA is primarily considered a biomarker for the diagnosis of prostate cancer, it is also used in the diagnosis of BPH because it is organ-specific rather than cancer-specific. It has been observed that some patients are diagnosed with both prostate cancer and BPH in the gray zone, which is the range where the serum PSA value is 4–10 ng/mL. Agnihatri et al. (13) reported that t-PSA values in the range of 4-20 ng/mL in symptomatic men have very limited value in reducing unnecessary biopsies, and the use of the f-PSA/t-PSA ratio is more beneficial. This ratio helps differentiate between benign conditions and malignancy, providing clinicians with better insights for decision-making. Consequently, implementing this approach could lead to more targeted interventions and improved patient outcomes. Additionally, utilizing the f-PSA/t-PSA ratio may also decrease the emotional and financial burden on patients by reducing the number of unnecessary procedures. As research continues to evolve, further refinements in PSA testing methodologies are likely to enhance the accuracy of prostate cancer diagnostics. These advancements will not only improve early detection rates but also facilitate more personalized treatment plans tailored to individual patient profiles. Ultimately, this progress underscores the importance of ongoing innovation in medical testing and the need for continuous education among healthcare providers. By fostering a deeper understanding of testing protocols and patient needs, healthcare professionals can significantly improve outcomes. As a result, patients will benefit from more effective care strategies that prioritize their health and well-being. One of the most commonly used tests to differentiate BPH and prostate cancer in prostate diseases is the f-PSA/t-PSA ratio. An increase in this ratio may be evaluated in favor of BPH. In the present study, t-PSA levels were similar between the groups. However, both f-PSA and the f-PSA/t-PSA ratio were significantly elevated in the BPH group compared to the controls. The comparable t-PSA values across the groups indicate that t-PSA may have limited diagnostic accuracy in patients with levels < 3 ng/mL. Our study revealed that BPH does not cause significant changes in GLP-1 levels. The current literature lacks sufficient investigations focusing on alterations in serum GLP-1 levels in BPH and the underlying mechanisms, if any changes occur. Most existing research in this area primarily examines the effects of GLP-1 and its analogues on prostate cancer patients. Experimentally, exendin-4, a GLP-1 receptor mimic, was administered to rats with BPH fed a high-fat diet for 12 weeks, and at the end of the study, increased GLP-1 receptor expression in prostate tissue was observed, but no increase in prostate size was noted. Another study demonstrated that blocking integrin receptors significantly alleviated prostatic hyperplasia in C57BL/6 mice. Recently, we found increased serum GLP-1 levels increased in both BPH and prostate cancer groups compared to healthy controls; however, the diagnostic value of GLP-1 for prostate cancer has limited diagnostic usefulness (with a sensitivity of 18% and specificity of 71%). Although the present study's

results showed an increase in GLP-1 in patients diagnosed with BPH with a t-PSA level of 3 ng/mL compared to healthy controls, this increase was not statistically significant. Given the limited number of studies on this topic and the predominance of research focusing on patients with prostate cancer, the diagnostic value of GLP-1 in BPH warrants further investigation. Gal-3 acts as a proteolytic substrate for PSA in human seminal plasma (5). Changes in Gal-3 expression are frequently observed in both malignant and premalignant conditions (18). Nevertheless, the exact molecular pathways governing the expression of Gal-3 in tumor cells are not fully understood (19). Gal-3 plays an important role in the pathogenesis of BPH, and studies suggest that it can be used as a promising potential therapeutic agent for the treatment of the disease (20). Similarly, serum Gal-3 levels were significantly elevated in individuals with BPH in the present study. This disparity may be attributed to the distinct anatomical origins and secretion mechanisms of Gal-3 and PSA within the prostate epithelium. Specifically, in prostate cancer, PSA is predominantly secreted by luminal epithelial cells, whereas Gal-3 is secreted by basal cells, reflecting differences in both cellular sources and secretion pathways (21). Although our study results did not show a significant correlation between the levels of PSA and Gal-3, the results of other studies showed findings contrary to this. However, most of these studies were performed on patients diagnosed with prostate cancer (22). There are no studies in the literature examining serum Gal-3 levels in BPH; therefore, we believe that whether Gal-3 will serve as a complementary biomarker to the PSA test in BPH or prostate cancer can be determined by further studies. The homeostasis of essential minerals is critical for maintaining normal organ function. Calcium (Ca) and magnesium (Mg) are two key elements involved in cellular growth and division. Beyond their fundamental roles, magnesium contributes to the stabilization of cell membranes, and its deficiency increases membrane permeability and vulnerability to oxidative stress, resulting in various cellular abnormalities (23). A significant relationship has been reported between serum Mg levels, the Ca/Mg ratio, and prostate cancer (24). In our study, no significant difference was observed in serum Ca levels between the control and BPH groups; however, in line with the literature, individuals with BPH had lower serum Mg levels than healthy individuals. This finding suggests that Mg supplementation may be beneficial in the management of BPH symptoms.

Study limitations: The limitation of this study is that BPH patients with t-PSA levels between 4 and 10 ng/mL, referred to as the gray zone in general routine practice, were not included as a separate group in the study, and the diagnostic significance of these two parameters was not investigated.

Conclusion

The findings of our investigation indicate that Gal-3 may play a substantial role in the genesis of BPH in patients over 40 years of age with a total PSA level below 3.0 ng/mL. It also revealed that the free PSA value is more valuable than the total PSA value in the diagnosis of BPH. This is significant as it is the first study to investigate the importance of Gal-3 and GLP-1 together in patients diagnosed with BPH. Our study should be supported by research that includes more participants, particularly those with PSA levels in the gray zone.

Ethical approval: This study was reviewed and approved by the Institutional Review Board at the University of Ordu and was performed under the principles of the Declaration of Helsinki. Ethics committee approval was received from the Ordu University Local Ethical Committee Under Reference No: 2022/278.

Conflict of interest: The authors have no conflict of interest.

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Author contributions: Concept (TN, BY, EB), Design (TN, BY, EB), Data Collection and/or Processing (TN, BY, EB), Analysis and/or Interpretation (TN, EB)

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