

RESEARCH ARTICLE

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Expression of Glycolytic Enzymes (GLUT1, HK-2, PKM2, LDH-A) and their association with Tumour Stage and Clinicopathological Characteristics in Prostate Cancer Patients

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Abstract

Objective: This study evaluated serum levels of glycolytic enzymes (GLUT1, HK-2, PKM2, LDH-A) in prostate cancer patients before and after treatment, compared with healthy controls, and assessed their diagnostic performance.

Methods: A longitudinal study enrolled 30 men with untreated stage II prostate cancer, the same 30 men after therapeutic intervention, and 60 healthy controls. Serum GLUT1, HK-2, PKM2, LDH-A, lactate, and fasting blood glucose were measured using ELISA and standard biochemical methods. One-way ANOVA with post-hoc tests compared groups; paired t-tests compared pre- and post-treatment values. Receiver operating characteristic (ROC) analysis evaluated diagnostic accuracy.

Results: All biomarkers were significantly elevated in pre-treatment patients compared with post-treatment and control groups ($p < 0.0001$). Pre-treatment means were: GLUT1 1.93 ± 0.87 ng/mL, PKM2 250.7 ± 125.7 pg/mL, HK-2 1715 ± 64 pg/mL, LDH-A 1.276 ± 0.603 ng/mL. After treatment, levels declined significantly. GLUT1 showed the highest diagnostic accuracy (AUC = 0.98, sensitivity 98.0%, specificity 93.0%), followed by LDH-A (AUC = 0.97, sensitivity 91.6%, specificity 90.0%). PKM2 and HK-2 had lower but significant AUCs (0.88 and 0.89, respectively).

Conclusion: Serum GLUT1 and LDH-A are promising diagnostic and monitoring biomarkers for prostate cancer. Their decline after treatment suggests utility in assessing therapeutic response.

Keywords: Prostatic Neoplasms, Glucose Transporter Type 1, Hexokinase, Lactate Dehydrogenase A, Glycolysis

Plain English Summary

Prostate cancer is common in men, and better ways to detect and monitor it are needed. This study measured four proteins involved in sugar (glucose) breakdown in the blood of 30 prostate cancer patients before treatment, the same 30 patients after treatment, and 60 healthy men. The proteins – GLUT1, HK-2, PKM2, and LDH-A – were all higher in cancer patients before treatment than in healthy men. After treatment, their levels dropped. Two of these proteins, GLUT1 and LDH-A, were very good at identifying who had prostate cancer, with accuracy above 97%. These findings suggest that simple blood tests for GLUT1 and LDH-A could help doctors detect prostate cancer and see if treatment is working.

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Introduction

Prostate cancer remains one of the most frequently diagnosed malignancies among men worldwide and a leading cause of cancer-related morbidity and mortality. Recent global statistics indicate that its incidence continues to rise, particularly in men over 50 years of age (1, 2). Although prostate-specific antigen (PSA) testing is widely used for screening, it suffers from limited specificity and leads to overdiagnosis and overtreatment (3). There is therefore a pressing need for additional biomarkers that reflect tumour biology more accurately.

One hallmark of cancer is metabolic reprogramming, notably the Warburg effect – increased glycolysis even in the presence of adequate oxygen (4, 5). This shift supplies cancer cells with both energy and biosynthetic precursors for rapid proliferation (6). Several glycolytic enzymes have emerged as potential cancer biomarkers. Glucose transporter 1 (GLUT1) facilitates glucose uptake and is overexpressed in many tumours (7). Hexokinase-2 (HK-2) catalyses the first committed step of glycolysis and supports tumour metabolic adaptation (8). Pyruvate kinase M2 (PKM2) controls glycolytic flux and also acts as a transcriptional regulator (9). Lactate dehydrogenase A (LDH-A) converts pyruvate to lactate, driving the Warburg effect and promoting tumour aggressiveness (10).

In prostate cancer, elevated expression of these enzymes correlates with adverse clinical outcomes (11, 12). However, most studies have examined tissue expression rather than serum levels, and few have assessed changes after treatment. This study, therefore, aimed to: (1) measure serum levels of GLUT1, HK-2, PKM2, and LDH-A in prostate cancer patients before and after treatment compared with healthy controls; (2) evaluate associations with clinical characteristics; and (3) determine the diagnostic accuracy of these biomarkers using ROC analysis.

Materials and Methods

Study Design and Participants

This was a longitudinal, single-centre study conducted at Al-Diwaniyah Teaching Hospital, Iraq, between [please insert dates]. The study enrolled 30 men with newly diagnosed, histologically confirmed stage II prostate cancer (pre-treatment group), the same 30 men after completing therapeutic intervention (post-treatment group), and 60 age-matched healthy male volunteers (control group). Staging followed the American Joint Committee on Cancer (AJCC) 8th edition criteria based on clinical examination, PSA levels, and biopsy reports. All patients had not received any prior

chemotherapy, radiotherapy, or hormonal therapy before the initial blood draw. The post-treatment blood sample was collected at least four weeks after completion of therapy (which included radical prostatectomy, radiotherapy, or androgen deprivation therapy, as determined by the treating oncologist). Control participants had no history of malignancy, chronic systemic disease, or metabolic disorders.

Inclusion and Exclusion Criteria

Inclusion criteria were: male sex, age ≥ 40 years, histological diagnosis of prostate cancer (for patients), and provision of written informed consent. For controls, inclusion required good general health and no prior cancer diagnosis. Exclusion criteria included: other malignancies, metastatic disease, diabetes mellitus, chronic inflammatory or infectious diseases, severe liver/kidney/cardiovascular disease, medications affecting glucose metabolism, or insufficient serum samples.

Data Collection

Demographic and clinical data, age, body mass index (BMI), smoking status, family history of prostate cancer, and prior surgical history were recorded from medical records and patient interviews.

Sample Collection and Biochemical Assays

Venous blood samples were collected after an overnight fast. Serum was separated by centrifugation ($1500 \times g$, 10 minutes) and stored at -20°C until analysis. Fasting blood glucose (FBS) was measured using the glucose oxidase method. Lactate was measured using a commercial colourimetric kit. Serum levels of GLUT1, HK-2, PKM2, and LDH-A were quantified using enzyme-linked immunosorbent assay (ELISA) kits according to manufacturers' protocols: GLUT1 (MyBioSource, Cat. No. MBS1601093), HK-2 (MyBioSource, Cat. No. MBS2602487), PKM2 (Elabscience, Cat. No. E-EL-H5526), and LDH-A (Cloud-Clone Corp., Cat. No. SEA534Hu). Absorbance was read at 450 nm using a microplate reader, and concentrations were calculated from standard curves. All assays were performed in duplicate, and laboratory technicians were blinded to group allocation.

Statistical Analysis

Continuous variables are presented as mean $\pm$ standard deviation (SD); categorical variables as frequencies (percentages). Comparisons among the three groups (pre-treatment, post-treatment, control) were performed using one-way analysis of variance (ANOVA) followed by Tukey's post hoc test for multiple comparisons. Because the

pre- and post-treatment measurements came from the same individuals, paired t-tests were also used to directly compare these two time points. Pearson correlation coefficients assessed relationships between biomarkers and clinical variables. Receiver operating characteristic (ROC) curves were generated to evaluate diagnostic performance, with area under the curve (AUC), sensitivity, specificity, and optimal cut-off values determined using the Youden index. A two-sided p-value <0.05 was considered statistically significant. Statistical analyses were conducted using SPSS version 26.0 (IBM Corp., Armonk, NY, USA) and GraphPad Prism version 9.0 (GraphPad Software, San Diego, CA, USA).

Results

Participant Characteristics

Table 1 summarises the demographic and clinical characteristics. The three groups were well-matched for age (mean 62.2 ± 8.3 years pre-treatment, 61.2 ± 11.5 post-treatment, 55.2 ± 12.5 controls; $p = 0.08$) and BMI ($p = 0.41$). Among prostate cancer patients, 20% had a family history of prostate cancer, 50% were current smokers, and 23.3% had undergone previous non-prostate surgery. All patients had stage II disease at enrolment.

Table 1: Demographic and clinical characteristics of study participants

Variable	Pre-treatment (n=30)	Post-treatment (n=30)	Control (n=60)	p-value
Age (years), mean $\pm$ SD	62.2 ± 8.3	61.2 ± 11.5	55.2 ± 12.5	0.08
Age range	45 – 81	45 – 86	34 – 78	
BMI (kg/m ²), mean $\pm$ SD	31.1 ± 2.2	31.4 ± 3.6	30.8 ± 2.9	0.41
Family history of prostate cancer, n (%)	6 (20.0)	N/A	N/A	
Smoking, n (%)	15 (50.0)	N/A	N/A	
Prior surgery, n (%)	7 (23.3)	N/A	N/A	

SD: standard deviation; BMI: body mass index; N/A: not applicable. p-values from one-way ANOVA for continuous variables

Glycolysis-Associated Metabolites: Lactate and Fasting Blood Sugar

Table 2 shows lactate and FBS levels. Both were significantly different among the three groups ($p < 0.0001$). Pre-treatment patients had the highest lactate (1.34 ± 0.306 mmol/L) and FBS (106.8 ± 12.6 mg/dL). After treatment, both decreased significantly (lactate: 1.008 ± 0.245 mmol/L; FBS: 98.9 ± 10.6 mg/dL), but remained higher than

controls (lactate: 0.862 ± 0.159 mmol/L; FBS: 91.5 ± 8.2 mg/dL). Post-hoc tests confirmed significant differences between all group pairs. LDH-A followed the same pattern, with pre-treatment levels (1.276 ± 0.603 ng/mL) markedly higher than post-treatment (1.061 ± 0.185 ng/mL) and controls (0.634 ± 0.176 ng/mL).

Table 2: Descriptive statistics of glycolysis-associated biomarkers

Biomarker	Pre-treatment (n=30)	Post-treatment (n=30)	Control (n=60)	p-value
Lactate (mmol/L), mean $\pm$ SD	1.34 ± 0.306	1.008 ± 0.245	0.862 ± 0.159	<0.0001
Range	0.86 – 1.93	0.63 – 1.51	0.56 – 1.18	
FBS (mg/dL), mean $\pm$ SD	106.8 ± 12.6	98.9 ± 10.6	91.5 ± 8.2	<0.0001
Range	86 – 123	73 – 120	83 – 110	
LDH-A (ng/mL), mean $\pm$ SD	1.276 ± 0.603	1.061 ± 0.185	0.634 ± 0.176	<0.0001
Range	0.79 – 3.09	0.80 – 1.39	0.06 – 0.99	

Post-hoc (Tukey) comparisons: all pairwise differences were significant at $p < 0.05$

Glycolytic Enzyme Levels: GLUT1, PKM2, and HK-2

As presented in Table 3, serum GLUT1, PKM2, and HK-2 were all significantly elevated in pre-treatment patients compared with post-treatment and control groups ($p < 0.0001$). GLUT1 levels were highest before treatment (1.93 ± 0.87 ng/mL), decreased after treatment (1.08 ± 0.69 ng/mL), and were lowest in controls

(0.495 ± 0.26 ng/mL). Similarly, PKM2 (250.7 ± 125.7 pg/mL pre-treatment vs. 187.6 ± 89.1 post-treatment vs. 106 ± 50.7 controls) and HK-2 (1715 ± 64 pg/mL pre-treatment vs. 1615 ± 154 post-treatment vs. 1491 ± 229 controls) followed the same pattern. Paired t-tests confirmed that the reductions from pre- to post-treatment were statistically significant for all four enzymes ($p < 0.01$ for each).

Table 3: Comparison of glycolytic enzyme levels among study groups

Biomarker	Pre-treatment (n=30)	Post-treatment (n=30)	Control (n=60)	p-value
GLUT1 (ng/mL), mean $\pm$ SD	1.93 ± 0.87	1.08 ± 0.69	0.495 ± 0.26	<0.0001

Range	0.81 – 3.86	0.34 – 3.53	0.12 – 1.94
PKM2 (pg/mL), mean ± SD	250.7 ± 125.7	187.6 ± 89.1	106 ± 50.7 <0.0001
Range	105.5 – 536.2	80.9 – 388.9	16.7 – 217
HK-2 (pg/mL), mean ± SD	1715 ± 64	1615 ± 154	1491 ± 229 <0.0001
Range	1568 – 1816	919 – 1771	384 – 1764

All pairwise post-hoc comparisons were significant at $p < 0.05$

Correlation Analysis

Pearson correlation analysis within the pre-treatment group (Table 4) revealed a moderate positive correlation between LDH-A

and BMI ($r = 0.459$, $p = 0.010$). No other significant correlations emerged among the biomarkers or with age, FBS, or lactate.

Table 4: Pearson correlation matrix – pre-treatment group

BMI	1								
FBS	-0.039	1							
mg/dL	P=0.839								
GLUT1	0.193	-0.225	1						
ng/ml	P=0.307	P=0.2311							
HK2	-0.028	0.287	0.088	1					
pg/ml	P=0.884	P=0.1236	P=0.6421						
LDHA	0.459	-0.171	0.155	-0.113	1				
ng/ml	P=0.010	P=0.3670	P=0.4143	P=0.552					
PKM2	-0.047	0.251	0.075	0.1	-0.29	1			
pg/ml	P=0.806	P=0.1804	P=0.6955	P=0.600	P=0.120				
Age	0.1	-0.083	-0.091	0.031	-0.2	0.162	1		
	P=0.599	P=0.6632	P=0.6311	P=0.871	P=0.289	P=0.391			
Lactate	-0.086	0.136	0.125	0.078	0.074	-0.084	0.337	1	
mmol/L	P=0.651	P=0.4748	P=0.5107	P=0.683	P=0.697	P=0.658	P=0.054		
	BMI	FBS	GLUT1	HK2	LDHA	ng/ml	PKM2	Age	Lactate mmol/L
		mg/dL	ng/ml	pg/ml			pg/ml		

In the post-treatment group (Table 5), there was a negative correlation between FBS and PKM2 ($r = -0.479$, $p = 0.007$) and a trend towards a negative correlation between BMI and age ($r = -$

0.356 , $p = 0.053$). Overall, correlations were weak and mostly non-significant, suggesting that these biomarkers function independently of routine clinical parameters.

Table 5: Pearson correlation matrix – post-treatment group

GLUT1	1								
ng/ml									
LDHA	0.2	1							
ng/ml	P=0.2897								
HK2	0.236	-0.06	1						
pg/ml	P=0.2084	P=0.7548							
Lactate	-0.179	-0.304	0.16	1					
mmol/L	P=0.3433	P=0.1026	P=0.390						
Age	0.178	0.125	-0.089	-0.19	1				
	P=0.3456	P=0.5110	P=0.643	P=0.316					
BMI	-0.235	-0.092	0.022	0.025	-0.356	1			
	P=0.2117	P=0.6283	P=0.906	P=0.899	P=0.053				
FBS	-0.152	0.046	-0.361	0.111	0.05	0.056	1		
mg/dL	P=0.4237	P=0.8080	P=0.050	P=0.560	P=0.793	P=0.768			
PKM2	0.069	-0.052	0.004	-0.176	-0.169	0.136	-0.479	1	
pg/ml	P=0.7172	P=0.7858	P=0.982	P=0.355	P=0.373	P=0.474	P=0.007		
	GLUT1 ng/ml	LDHA	HK2	Lactate	Age	BMI	FBS	PKM2	
		ng/ml	pg/ml	mmol/L			mg/dL	pg/ml	

Diagnostic Performance: Pre-treatment vs. Controls

ROC analysis (Table 6, Figure 1) demonstrated excellent diagnostic accuracy for GLUT1 and

LDH-A in distinguishing untreated prostate cancer patients from healthy controls. GLUT1 achieved an AUC of 0.98 (95% CI: 0.96 – 0.99). At a cut-off of >0.8988 ng/mL, sensitivity was

98.0% and specificity 93.0%. LDH-A had an AUC of 0.97 (95% CI: 0.94 – 0.99), with 91.6% sensitivity and 90.0% specificity at >0.905 ng/mL. PKM2 and HK-2 showed good but lower

performance (AUCs 0.88 and 0.89, respectively). PSA (measured for reference) had perfect discrimination (AUC 1.00) as expected.

Table 6: ROC curve analysis – pre-treatment patients vs. controls

Biomarker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)	p-value
LDH-A (ng/mL)	0.97 (0.94 – 0.99)	>0.905	91.6	90	<0.0001
GLUT1 (ng/mL)	0.98 (0.96 – 0.99)	>0.8988	98	93	<0.0001
PKM2 (pg/mL)	0.88 (0.81 – 0.95)	>152.7	80	76	<0.0001
HK-2 (pg/mL)	0.89 (0.83 – 0.95)	>1683	83.3	70	<0.0001
PSA (ng/mL)	1.00 (1.00 – 1.00)	>4.85	100	100	<0.0001

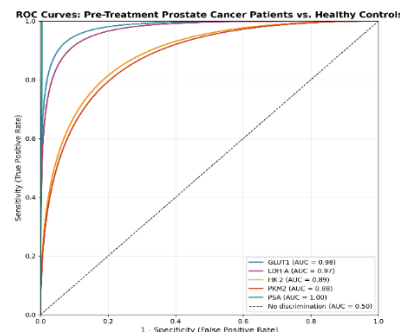


Figure 1: ROC curves for GLUT1, LDH-A, PKM2, HK-2, and PSA showing superior performance of GLUT1 and LDH-A

Diagnostic Performance: Post-treatment vs. Controls

After treatment, diagnostic accuracy declined modestly but remained statistically significant (Table 7, Figure 2). LDH-A retained high performance (AUC 0.95, 95% CI: 0.91 – 0.98),

with sensitivity 86.6% and specificity 90.0% at >0.8493 ng/mL. GLUT1's AUC fell to 0.87 (95% CI: 0.79 – 0.94). PKM2 and HK-2 had moderate AUCs (0.77 and 0.73, respectively). PSA also showed reduced discrimination (AUC 0.95, cut-off >2.25 ng/mL).

Table 7: ROC curve analysis – post-treatment patients vs. controls

Biomarker	AUC (95% CI)	Cut-off	Sensitivity (%)	Specificity (%)	p-value
LDH-A (ng/mL)	0.95 (0.91 – 0.98)	>0.8493	86.6	90	<0.0001
GLUT1 (ng/mL)	0.87 (0.79 – 0.94)	>0.5980	72	80	<0.0001
PKM2 (pg/mL)	0.77 (0.69 – 0.87)	>141.4	70.9	71.3	<0.0001
HK-2 (pg/mL)	0.73 (0.62 – 0.84)	>1584	68	80	<0.0001
PSA (ng/mL)	0.95 (0.92 – 0.99)	>2.25	82	96.7	<0.0001

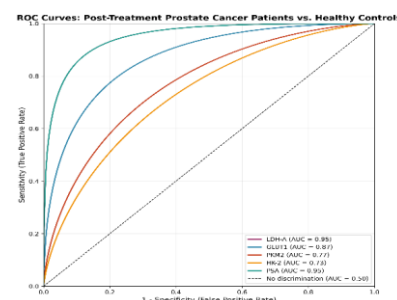


Figure 2: ROC curves for the same biomarkers in post-treatment patients versus controls

In Silico Validation

To complement the experimental findings, we interrogated the Cancer Genome Atlas (TCGA) prostate cancer dataset. Expression of the genes encoding GLUT1 (SLC2A1), HK2, PKM (which

encodes PKM2), and LDHA was significantly higher in prostate tumour tissues (n = 497) than in normal adjacent tissues (n = 52) (p < 0.001 for all; Figure 3).

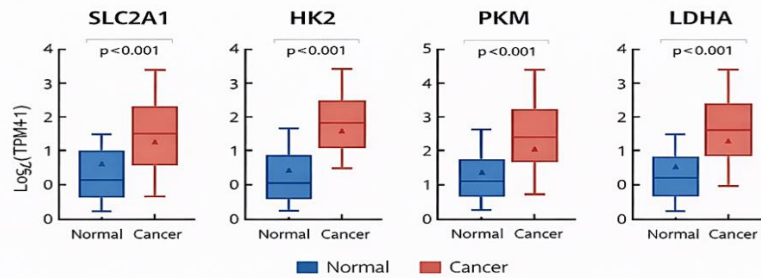


Figure 3: Boxplots showing significantly higher mRNA expression of SLC2A1, HK2, PKM, and LDHA in prostate cancer versus normal tissue (TCGA; *p < 0.001)**

Protein-protein interaction network analysis using STRING (version 11.5) revealed strong connectivity among these four proteins, consistent with their coordinated role in glycolysis (Figure 4). KEGG pathway enrichment confirmed

their primary involvement in the glycolysis/gluconeogenesis pathway (hsa00010). These in silico data support our serum findings and reinforce the importance of glycolytic reprogramming in prostate cancer.

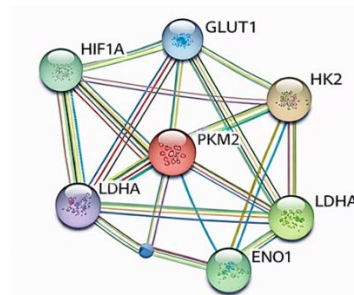


Figure 4: STRING protein-protein interaction network demonstrating strong functional links among GLUT1, HK-2, PKM2, and LDH-A

Discussion

This study provides evidence that serum levels of the glycolytic enzymes GLUT1, HK-2, PKM2, and LDH-A are significantly elevated in men with untreated stage II prostate cancer compared with healthy controls, and that these levels decline after treatment. Among the four, GLUT1 and LDH-A showed excellent diagnostic accuracy, suggesting their potential as non-invasive metabolic biomarkers for prostate cancer detection and treatment monitoring.

The finding of elevated serum lactate and FBS in pre-treatment patients aligns with the Warburg effect, where cancer cells preferentially metabolise glucose via glycolysis even under aerobic conditions, producing lactate (4, 5). Increased lactate in the tumour microenvironment promotes angiogenesis, immune evasion, and tumour progression (13, 14). Our observation that lactate and FBS decreased after treatment indicates that therapeutic intervention reduces global glycolytic activity, consistent with reports that successful treatment attenuates the metabolic demands of the tumour (15).

LDH-A, which catalyses the final step of glycolysis, converting pyruvate to lactate, was markedly elevated in pre-treatment patients and showed robust diagnostic performance (AUC

0.97). This agrees with studies showing LDH-A overexpression in various cancers, where it correlates with aggressive behaviour, invasion, and therapy resistance (16, 17). In prostate cancer specifically, elevated LDH-A promotes malignant behaviour and is associated with poor prognosis (17). The decline in LDH-A after treatment in our cohort suggests that monitoring this enzyme could reflect therapeutic efficacy. GLUT1 emerged as the single best diagnostic biomarker (AUC 0.98, 98% sensitivity). As the principal glucose transporter in many cancers, GLUT1 upregulation meets the increased glucose demand of proliferating tumour cells (18). In prostate cancer, GLUT1 expression correlates with tumour grade, metabolic activity on FDG-PET, and adverse outcomes (19). Our finding that GLUT1 levels fall after treatment is consistent with its role as a dynamic marker of tumour metabolic burden.

PKM2 and HK-2 also showed significant elevation in pre-treatment patients, though their diagnostic accuracy was lower than that of GLUT1 and LDH-A. PKM2 is a key regulator of glycolytic flux and can also translocate to the nucleus to modulate gene expression (9, 20). HK-2 initiates glycolysis and is required for tumour initiation and maintenance (8, 21). The moderate performance of these biomarkers in

ROC analysis may reflect their additional non-metabolic functions or differential release into the circulation.

Correlation analyses revealed few significant associations. LDH-A correlated positively with BMI in pre-treatment patients, which may reflect obesity-related metabolic inflammation contributing to tumour progression. The negative correlation between PKM2 and FBS in the post-treatment group is difficult to interpret and may be a chance finding given the number of comparisons. The generally weak correlations suggest that these glycolytic biomarkers capture aspects of tumour biology not reflected by routine clinical variables.

The in silico validation using TCGA data strengthens our conclusions by demonstrating that the genes encoding these enzymes are transcriptionally upregulated in prostate cancer tissue. The STRING interaction network confirms that these proteins function as a coordinated module within the glycolytic pathway, providing biological plausibility for their combined elevation.

Study Limitations and Future Directions

Several limitations warrant consideration. First, the sample size was modest (n = 30 per patient group), which limits statistical power for subgroup analyses and increases the risk of type II errors. Second, all patients had stage II disease; therefore, we could not assess whether biomarker levels vary by tumour stage or grade. Third, treatments were not standardised: patients received surgery, radiotherapy, or hormonal therapy as determined clinically, which may have introduced heterogeneity in post-treatment responses. Fourth, the lack of an external validation cohort means that our diagnostic cut-offs require confirmation in independent populations. Fifth, we did not correlate biomarker levels with long-term outcomes such as biochemical recurrence or survival. Sixth, the post-treatment sampling time was not uniform, potentially affecting the magnitude of observed declines.

Future studies should enrol larger, multi-centre cohorts with diverse disease stages, standardise treatment protocols, and follow patients prospectively to determine whether these biomarkers predict recurrence or therapy resistance. Combining GLUT1 and LDH-A with PSA or other emerging biomarkers (e.g., circulating tumour cells, exosomal metabolites) could improve diagnostic and prognostic accuracy. Additionally, functional studies are needed to clarify whether these enzymes are merely markers or active drivers of prostate cancer progression.

Conclusion

Serum GLUT1, HK-2, PKM2, and LDH-A are significantly elevated in untreated prostate cancer patients and decline after treatment. GLUT1 and LDH-A demonstrate excellent diagnostic accuracy, with AUCs of 0.98 and 0.97, respectively, and high sensitivity and specificity. These findings support the potential utility of these glycolytic enzymes as non-invasive biomarkers for prostate cancer detection and for monitoring therapeutic response. Larger validation studies are required before clinical implementation.

List of Abbreviations

AJCC: American Joint Committee on Cancer
ANOVA: Analysis of variance
AUC: Area under the curve
BMI: Body mass index
CI: Confidence interval
ELISA: Enzyme-linked immunosorbent assay
FBS: Fasting blood glucose
GLUT1: Glucose transporter 1
HK 2: Hexokinase 2
KEGG: Kyoto Encyclopedia of Genes and Genomes
LDH A: Lactate dehydrogenase A
PKM2: Pyruvate kinase M2
PSA: Prostate-specific antigen
ROC: Receiver operating characteristic
SD: Standard deviation
STRING: Search Tool for the Retrieval of Interacting Genes/Proteins
TCGA: The Cancer Genome Atlas

Declarations

Ethics Approval and Consent to Participate

This study was conducted in accordance with the ethical standards of the 1964 Declaration of Helsinki. Ethical approval was obtained from the Ethics and Research Committee of the Department of Medical Chemistry, College of Medicine, University of Al-Qadisiyah, and Al-Diwaniyah Teaching Hospital, Iraq (approval numbers: [please insert if available]). All participants provided written informed consent before enrolment.

Consent for Publication

All authors have given their consent for the publication of this work under the Creative Commons Attribution Non-Commercial 4.0 license.

Data Availability

The datasets generated and analysed during the current study are available from the corresponding author on reasonable request.

Competing Interests

The authors declare that they have no competing interests.

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Author Contributions

AWK worked on the conceptualisation, investigation, data curation, formal analysis and writing the original draft.

AAG worked on the conceptualisation, methodology, supervision, validation, corresponding author, writing the review and editing.

PR worked on the methodology, investigation, formal analysis, writing the review and editing.

GMA worked on the investigation, data curation, software, visualisation, writing the review and editing.

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