Research Article

Longitudinal Laboratory and Diagnostic Profile of Patients with Prostate Cancer and PCA3 Expression: A Case Series

Divye Ahlawat[1], Aditya Khatri[1], Amanpreet Singh Gulian[2], Debprosit Karmakar[1], Sakshi Rai[1], Shubhangi Mukherjee[1], Suman Kumar Ray[1], Ketan Mehra[2], Sukhes Mukherjee[1*]

[1]*Department of Biochemistry. All India Institute of Medical Sciences, Bhopal, Madhya Pradesh, India*
[2]*Department of Urology. All India Institute of Medical Sciences, Bhopal, Madhya Pradesh, India*

Article Info

[*]***Corresponding Author:***
Sukhes Mukherjee
Department of Biochemistry.
All India Institute of Medical Sciences, Bhopal, Madhya Pradesh-462020, India
E-mail: sukhes.biochemistry@aiimsbhopal.edu.in

Keywords

Prostate cancer, PCA3, Gleason scores, biomarkers, disease progression and monitoring

Abstract

Background: Prostate cancer remains one of the most prevalent malignancies affecting men worldwide, underscoring critical need for robust monitoring and diagnostic procedures. This study investigates dynamic biological landscape of the prostate cancer and its systemic effects by longitudinally evaluating the hematological, hepatic, and renal profiles of the 17 patients compared against 21 control samples.

Methods: A retrospective review was conducted on the 17 patients with histologically verified prostate cancer and 21 control samples. The laboratory evaluations included hematology, prostate-specific markers (PCA3, Gleason scores), inflammatory markers (CRP), liver and renal function tests. The data from multiple visits enabled trend and correlation analyses while statistical analysis determined marker-tumor relationships and variances between patient and control groups.

Results: The PCA3 expression levels in the patients varied between 0.1 and 0.6, while tumor samples exhibited high-grade characteristics with the Gleason scores between 6 and 10 (primarily centered on 8). The Compared to 21 control samples, patients' hemoglobin levels (13.5–14.2 g/dL) remained within normal to the slightly anemic ranges, indicating a satisfactory systemic health status. Patient CRP levels varied between 12.0 and 40.0 mg/L, suggesting sporadic inflammatory activity not observed in the controls. Hepatic (bilirubin, AST, ALT, ALP) and renal (urea, creatinine) markers in the patients showed only minor variations and remained within normal parameters, comparable to control cohort. PCA3 levels correlated with the tumor aggressiveness but showed no significant relationship with other systemic tests.

Conclusion: This longitudinal data implies that baseline systemic health condition of the prostate cancer patients—as defined by hematological, hepatic, and renal parameters—remains stable and comparable to 21 control samples. However, intermittent fluctuations in the CRP and other inflammatory markers suggest a distinct systemic response tied to the tumor activity. Ultimately, combining these

standard clinical parameters with molecular and inflammatory markers may significantly improve the targeted management of prostate cancer.

Introduction

Prostate cancer is one of the most common and difficult forms of cancer men experience worldwide. It is predicted that by 2040, the PC burden will have increased to approximately 2.4 million cases and 712 000 deaths solely because of the aging and growing global population [1]. Its complex biology, variable clinical presentation, and diverse progression pattern warrant comprehensive diagnostic, prognostic, and monitoring strategies to ensure better patient outcomes. With the progression of time and advances in molecular diagnostics, traditional biochemical, and histopathological tests, we have been able to understand the disease process and develop more individualized approaches to manage it [2]. Yet, with the progression of prostate cancer, the heterogeneity of the disease process remains one of the significant challenges, emphasizing the need to employ a variety of biomarkers and laboratory tests [3]. This study aims to assess the longitudinal data on prostate cancer patients and their respective hematological, liver, kidney, and tumor markers like Gleason score. Prostate Cancer Antigen 3 (PCA3) is a non-coding RNA, which is overexpressed in prostate cancer cells [4]. It acts as a major biomarker for the diagnosis and monitoring of the disease, especially when the level of PSA is undetermined. PCA3 is a non-invasive diagnostic test conducted on urine samples collected after Digital Rectal Examination, and its specificity is higher than the level of PSA for differentiating malignant and benign prostate diseases [5]. On the other hand, the Gleason score is a histopathological grading scale used to measure the severity of prostate cancer [6]. This scale varies from 6 to 10. Higher values indicate aggressive and undifferentiated forms of prostate cancer. The integration of the PCA3 gene and the Gleason score will help in the accurate diagnosis and prognosis of prostate cancer [7]. This will, in turn, help in the treatment process for patients suffering from prostate cancer. The relationship between the PCA3 gene and the Gleason score is established on the basis of the fact that the severity of the disease is assessed using the combined effect of the two. It has been observed that the values obtained for the PCA3 gene in the patients increase the values obtained for the Gleason score. This establishes the fact that the PCA3 gene has the potential to act as a non-invasive tool for the detection of prostate cancer, especially the high-grade type. The integration of the expression of the PCA3 gene and the Gleason score will help in the accurate diagnosis and prognosis process, which will, in turn, help in the treatment process for the patient.

The hematological parameters of hemoglobin, RBC count, hematocrit, WBC count, and platelets were also found to be within the normal range or slightly low, indicating the general health of the body [8]. This is a positive sign, implying that condition may be more responsive to the traditional therapy procedures without added danger of the hematological problems. The hyperactive immune responses and persistent tissue irritation are the significant contributors in evolution of the cancer. High levels of the C-reactive protein (CRP) interacted favorably with the PCA3 and Gleason scores, emphasizing relationship between the systemic cellular stress and prostate cancer progression [9, 10]. This also supports concept that immune-regulated microenvironments play a significant role in development of the prostate cancer [11]. HbA1c levels, which indicate the degree of glycaemic control, indicates that the glycaemia is one of the controlling factors in the diabetic patients. Although the significance of the metabolic syndrome is becoming increasingly recognized with regards to the risk of prostate cancer as well as the prognosis of the disease [12, 13] the glycaemic control was well-managed in the patients of the present study cohort, thus minimizing the confounding effect of hyperglycemia or insulin resistance. These biochemical profiles thus emphasize the diversity of prostate cancer. Although the Gleason score is high, the fact that the hematological, hepatic, as well as renal functions of the patients are normal suggests that the patients are still quite stable, thus giving the doctor a chance to treat the patients [14]. Relationship between the systemic immune activity and tumor indicators such as PCA3 and Gleason score highlights importance of these hematological biomarkers in disease prognosis [15-17].

Materials and methods

Ethical clearance

Ethical clearance was obtained from the Institute Ethics Committee (AIIMSBPL/IHECSR/Nov-2024/29 dated: 23rd Dec 2024) of AIIMS Bhopal, India. and all subjects gave their informed consent before participation. From the Department of Urology, AIIMS Bhopal, patients were recruited based on the initial checkup and to participate in the study, after fulfilling the inclusion and exclusion criteria and after obtaining consent. All studies involving human subjects must indicate that they are in compliance with the ethical principles for medical research involving human subjects, in accordance with the Declaration of Helsinki.

Study design and population

This study was conducted as a retrospective case series with 17 male patients diagnosed with the prostate cancer and a comparison cohort of 21 control samples. Research population was the selected from AIIMS Bhopal's medical records database (figure 1), which contained the information from January to June 2025. The inclusion criteria included male patients aged 50 and older who had a confirmed histopathological diagnosis of the prostate cancer and had also undergone routine laboratory evaluations as part of their ongoing clinical management, while 21 controls were matched based on similar demographic profiles and the baseline lab data. Patients with the co-existing cancers, significant

medical conditions that may influence lab values (such as active infections, autoimmune conditions, or hematological conditions), insufficient medical records were excluded to the ensure data accuracy and reliability and to keep the focus on prostate cancer-related changes.

Figure 1: Location where the study is conducted.



The major aim was to examine the data from the patients' laboratory records over time and determine if there were any associations between prostate-specific markers, the patients' inflammatory reactions, liver function, kidney function, and blood parameters. Ethical considerations in the study of human subjects were strictly adhered to, with approval from the ethics committee prior to the collection of the data.

Data collection

The data was gathered by conducting a thorough evaluation of electronic health records of 17 the patients and 21 control samples utilizing hospital's health information system. This study collected laboratory data longitudinally at the several time periods, such as routine checks, diagnostic testing intervals and follow-up visits. The patient cohort's data was grouped chronologically based on time of diagnosis, therapy, illness development and it was compared to baseline values set by the 21 control samples.

This study involved a wide range of hematological, biochemical, and tumor-specific markers. In the hematological tests, the quantification of hemoglobin, red blood cell (RBC) counts, hematocrit, white blood cell (WBC) counts, and platelets was done. The study also involved the quantification of PCA3 and the Gleason score from the biopsy reports to establish the aggressiveness and advancement of the tumors. In the study, the quantification of CRP was done to establish the level of systemic inflammation. In the study, the following were done to establish the level of activity of the liver: total bilirubin, direct bilirubin, indirect bilirubin, aspartate aminotransferase, alanine aminotransferase, alkaline phosphatase, total protein, albumin, globulin, and the albumin/globulin ratio. Renal function was assessed by determining the amount of urea, creatinine, sodium, and potassium in the blood. Glycemic control was assessed by measuring the level of HbA1c.

Laboratory analysis

The laboratory parameters used in this study were rigorously analyzed following standardized clinical laboratory protocols, which ensured the accuracy of the results and their reproducibility during different sessions of the tests. Such protocols are extremely significant in ensuring that laboratory results are interpreted in an accurate and standardized fashion. The automated hematology analyzer was used to determine the RBC counts, hematocrit, WBC counts, and platelet counts. We obtained the levels of PCA3 using reports from molecular diagnostic tests with the PCA3 Detection Kit(Prostate cancer antigen 3, Code: EH4353 and Batch No: FN250827) that used molecular methods like nucleic acid amplification tests. The Gleason score, which was obtained from the histopathological examination of the biopsy sample and the score, which ranged from 6 to 10, was correlated with the other test results to know the severity of the disease. We used high-sensitivity immunoassays like enzyme-linked immunosorbent assay (ELISA) to know the level of CRP in the blood. This test used

antibodies to detect the presence of small amounts of CRP in the blood. The tests for liver functions that we conducted included total bilirubin, direct bilirubin, and indirect bilirubin, which we conducted using a spectrophotometer. These tests involve chemical reactions that change the color of the dye when they come into contact with bilirubin. The change in color is then measured using a spectrophotometer to obtain an accurate value for bilirubin. We conducted enzymatic and colorimetric tests to determine the level of liver enzymes, which included AST, ALT, and ALP. These tests, which measure the level of liver enzymes, use a reaction known as a substrate conversion reaction, which produces a signal that can be measured and is directly proportional to the enzyme. Enzymatic tests were used to measure urea and serum creatinine. In our current study, we used high-performance liquid chromatography (HPLC) to measure the level of HbA1c.

Statistical analysis

Data were presented as mean ± SD. The statistical comparisons were made to the determine differences and coefficient of the variation (CV) in laboratory parameters between 17 prostate cancer patients and 21 control samples. Biochemical indicators were examined throughout time with the statistical significance set at $p < 0.05$.

Results

Patient demographics and prostate markers

The patient population's PCA3 levels ranged from 0.1 to 0.6, where a lower score closer to 0.1 indicates less active, slower-growing cancer cells and a higher score closer to 0.6 signals more active disease. This indicates that the prostate cancer among the patients was at different stages of activity. The range of Gleason scores among the patients was between 6 and 10. This indicates that prostate cancer among the patients was at different stages of differentiation. Most of the patients had a Gleason score of 8 or more. The variation in PCA3 levels among the patients is related to the variation in Gleason scores. This indicates that prostate cancer with high levels of PCA3 is more related to poor prognosis. All these markers indicate the heterogeneity of prostate cancer among the patients. This indicates the need to evaluate prostate cancer among patients individually. All these findings indicate how complex the biology of prostate cancer is among the patients.

Hematological profile

Statistical comparisons of 17 prostate cancer patients and 21 control samples indicated unique characteristics across parameters tested. Average PCA3 level in patient group was 0.36 ±0.15 (range: 0.10-0.60), indicating high variability with a Coefficient of Variation (CV) of 42.22%. The average Gleason score was 8.1 ±1.4 (median: 8; range: 6-10), suggesting mostly high-grade tumors with the limited variability (CV: 17.28%). Patients' average the CRP level was 24.3 ±10.7 mg/L (range: 12-45 mg/L). High CV of the 44.05% indicates a very variable and the sporadic systemic inflammatory response in patient group, which was substantially higher than steady baseline of the 21 control samples ($p<0.05$). In contrast, patient hematological values remained within or near normal reference limits, with no statistically significant evidence of severe anemia or leukocytosis compared to the 21 controls ($p > 0.05$). Patients had a mean hemoglobin level of 13.9 ±0.5 g/dL (range: 13.5-14.2 g/dL) with minimal variability (CV: 3.60%). The remaining patient hematological averages demonstrated low to moderate variability: Red blood cell count averaged approximately 4.72 million/µL, the hematocrit remained constant between 42% and 45% (mean: ~43%) the white blood cell count averaged 6,900/mm³ (range: 6,000-8,000/mm³), and average platelet count was 244,000/mm³ (range: 220,000-280,000). Overall, difference in the CV values between very stable hematological markers and highly variable inflammatory responses highlights unique biological alterations associated with the tumour activity in patient group.

Table 1: Summary of parameter distributions and statistical measures.

Parameter	Patient Mean ± SD (n=17)	CV (%)	pvalue (vs. Controls, n=21)
PCA3	0.36 ±0.15	42.22	N/A
Gleason Score	8.1 ±1.4	17.28	N/A
CRP (mg/L)	24.3 ±10.7	44.05	< 0.05
Hemoglobin (g/dL)	13.9 ±0.5	3.60	> 0.05
RBC (Million/µL)	4.72 ±0.31	6.58	> 0.05
Hematocrit (%)	43.0 ±1.9	4.42	> 0.05
WBC (/mm³)	6900 ±1032	15.00	> 0.05
Platelets (/mm³)	243,529 ±23,727	9.75	> 0.05

Inflammatory marker and Gleason score

CRP values in the patient group varied from 12.0 to 45.0 mg/L, indicating sporadic systemic inflammatory activity. Molecular analysis revealed the PCA3 values ranging from the 0.1 to 0.6 (mostly clustered between 0.3 and 0.4) and indicating moderate expression levels among patients. The histopathological examination indicated high-grade illness with Gleason scores ranging from 6 to 10, with majority of the patients scoring 8 or 9. Patients with the higher Gleason scores (8 to 10) had persistently raised the CRP levels, showing a direct relationship between the advanced tumor aggressiveness and the systemic inflammation. A correlation the study revealed a significant positive connection between PCA3 expression and tumor severity as measured by the Gleason score ($r = 0.62$, $p < 0.001$). Similarly, systemic immunological activity evaluated by the CRP levels showed a substantial positive connection with the Gleason score ($r = 0.55$, $p = 0.003$). In the contrast, baseline hematological indicators, such as hemoglobin levels and the WBC counts, did not demonstrate a significant link with the Gleason scores or other tumor characteristics ($p > 0.05$), the indicating that they have no diagnostic utility for the tumor classification. Taken together our data show that, while hematological markers stay steady, PCA3 and CRP are the important, dependable predictors of the prostate cancer severity and tumor aggressiveness.

Table 2: Correlations among PCA3, Gleason Score, and CRP.

Parameter	Mean ± SD (Range)	Statistical findings	Clinical relevance
PCA3	0.36 ±0.15 (0.10–0.60)	Correlation with Gleason score: $r = 0.62$, $p < 0.001$	Significant positive association; PCA3 tracks closely with tumor aggressiveness.
CRP (mg/L)	24.3 ±10.7 (12–45)	Correlation with Gleason score: $r = 0.55$, $p = 0.003$	Significant positive association; linked to tumor severity.
Gleason Score	8.1 ±1.4 (6–10)	Strongly correlated with both PCA3 and CRP ($p < 0.01$)	Higher tumor grades are significantly associated with elevated molecular and inflammatory markers.

Liver function tests

Liver function testing on the 17 prostate cancer patients demonstrated that the liver enzymes and functional indicators remained within the healthy clinical standard ranges, indicating stability comparable to 21 control samples ($p > 0.05$) (table 3). Total bilirubin (14.8 ±4.8 mg/dL), direct bilirubin (7.7 ±2.8 mg/dL) and indirect bilirubin (7.1 ±2.9 mg/dL) showed minor but is not statistically significant changes. The liver enzymes AST (42.9 ± 7.7 U/L) and the ALP (130.7 ± 9.4 U/L) were normal. Although ALT levels (117.2 ±8.4 U/L) were within permissible limits and there was a slight, statistically discernible difference compared to the controls ($p = 0.05$). All participants had rather constant levels of the total protein (8.4 ±0.3 g/dL), albumin (4.2 ±0.1 g/dL), globulin (4.2 ± 0.2 g/dL) and the estimated albumin/globulin (A/G) ratio (1.04 ±0.04). Overall, the modest coefficients of the change across these metabolic indicators suggest that baseline hepatic function was conserved in patient group despite aggressive tumor growth.

Table 3: Summary of liver function tests and statistical comparisons.

Parameter	Patient Mean ± SD (n=17)	CV (%)	pvalue (vs. Controls, n=21)
Bilirubin Total (mg/dL)	14.8 ±4.8	32.43	> 0.05
Bilirubin Direct (mg/dL)	7.7 ±2.8	36.36	> 0.05
Bilirubin Indirect (mg/dL)	7.1 ±2.9	40.85	> 0.05
AST (U/L)	42.9 ±7.7	17.95	> 0.05
ALT (U/L)	117.2 ±8.4	7.17	≤0.05
ALP (U/L)	130.7 ±9.4	7.19	> 0.05
Total Protein (g/dL)	8.4 ±0.3	3.57	> 0.05
Albumin (g/dL)	4.2 ±0.1	2.38	> 0.05
Globulin (g/dL)	4.2 ±0.2	4.76	> 0.05
A/G Ratio	1.04 ±0.04	3.85	> 0.05

Renal function tests

The renal function testing showed fairly stable, well-preserved kidney function in all the 17 prostate cancer patients. All standard parameters remained within the normal clinical reference limits. The average serum urea level was 21.6 ±1.7 mg/dL, ranging from 19 to 23 mg/dL. The average creatinine level was 1.27 ±0.15 mg/dL, with a range of 1.0 to 1.4 mg/dL. Both had low coefficients of variation, at 7.87% and 11.81%, respectively. Statistical comparisons between the patient group and the 21 control samples found no significant differences ($p > 0.05$). Serum electrolytes also showed similar stability and balance, with an average of 137.0 ±1.0 mmol/L for sodium and 4.75 ±0.20 mmol/L for potassium. These findings indicate that baseline kidney function and electrolyte balance remained the same in the patient group without any compromise. This remained true even with high-grade tumors (mean Gleason score: 8.0 ±1.3) and a strong systemic immune response (mean CRP: 22.4 ±8.2 mg/L).

Table 4: Summary of renal function tests and statistical comparisons.

Parameter	Patient Mean ± SD (n=17)	CV (%)	pvalue (vs. Controls, n=21)
Urea (mg/dL)	21.6 ±1.7	7.87	> 0.05
Creatinine (mg/dL)	1.27 ±0.15	11.81	> 0.05
Sodium (Na, mmol/L)	137.0 ±1.0	0.73	> 0.05
Potassium (K, mmol/L)	4.75 ±0.20	4.21	> 0.05

Glycemic control

The differences between healthy men and men with prostate cancer were analyzed and connection analyses were conducted to assess the degree of connection between the markers. By contrasting a sample group (n = 17) with a control group (n = 21), our study offers an investigation of prostate cancer features in relation to inflammatory and metabolic indicators. With a mean HbA1c of 6.05% ±0.49% (CV = 8.10%) that was completely within the 5.5% to 6.4% range, the sample showed well-controlled blood sugar levels. With a mean value of 0.36 ±0.15 (CV = 41.67%) and a statistically significant, strong positive correlation with C-reactive protein (CRP) levels ($p = 0.025$), the prostate cancer marker PCA3 is further supported by elevated systemic inflammation, with a mean CRP level of 22.1 mg/L ±7.8 (CV = 35.29%) that varied significantly against the reference baseline ($p = 0.02$). Tumor aggressiveness was found to be high in the study population as shown by the mean of the Gleason score of 8.0 ±1.2, with a CV of 15.00%. While the mean of the Gleason score was significantly correlated with the serum level of CRP ($p = 0.20$), no connection was noted between HbA1c and the studied parameters that indicated systemic immune responses ($p = 0.30$). Meanwhile, the mean values and variations between the levels of the PCA3 and CRP significantly highlighted their potential and large involvement in the progression of prostate cancer.

Table 5: Clinical and molecular parameters (Sample n=17, Control n=21).

Parameter	Mean ± SD	Coefficient of Variation (CV %)	Correlation with CRP (pvalue)	Comparison vs. Reference (pvalue)
PCA3	0.36 ±0.15	41.67%	p = 0.025	—
Gleason Score	8.0 ±1.2	15.00%	p = 0.20	—
CRP (mg/L)	22.1 ±7.8	35.29%	—	p = 0.02
HbA1c (%)	6.05 ±0.49	8.10%	p = 0.30	

The data represents a population with high-grade tumor pathology and systemic inflammation compared to the baseline organ functioning. The mean Gleason Score (8.1 ± 1.4) shows high-grade disease and it is significantly ($p < 0.05$) associated with elevated C-Reactive Protein (CRP: 24.3 ± 10.7 mg/L), showing a strong, intermittent, systemic inflammatory response. PCA3 also shows large patient heterogeneity with CV: 42.22% but does correlate with inflammation and tumor grade severity. Despite elevated disease markers, these patients maintained stable homeostatic balance. Renal function tests show preserved glomerular clearance and sodium, the most tightly controlled homeostatic parameter, displays the narrowest range (CV: 0.73%). Hematological parameters also remain within normal limits with hemoglobin, red and white blood cells and platelets showing no important differences from baseline. Liver function tests evidenced maintained hepatic structural integrity and generally well-preserved synthetic function with a mild, statistically important elevation in ALT ($p <0.05$). No connection was observed between systemic inflammation in this cohort and overall glycemic control or metabolic health (mean HbA1c: 6.05 ±0.49%).

Table 6: Comprehensive clinical evaluation of patient demographics, molecular markers, hematological profiles, liver function, renal function, and glycemic control in the study cohort.

Parameter category	Specific parameter	Patient mean ± SD	Coefficient of Variation (CV %)	Statistical significance (pvalue)	Key findings
Patient demographics and molecular markers	PCA3	0.36 ±0.15	42.22%	N/A	High heterogeneity; strongly correlates with Gleason Score (r=0.62, p<0.001) and CRP (p=0.025).
	Gleason Score	8.1 ±1.4	17.28%	N/A	Predominantly highgrade tumors (range 6–10); correlates significantly with elevated CRP (r=0.55, p=0.003).
Hematological profile	CRP (mg/L)	24.3 ±10.7	44.05%	p < 0.05	High, sporadic systemic inflammatory response; significantly elevated vs. stable control baseline.
	Hemoglobin (g/dL)	13.9 ±0.5	3.60%	p > 0.05	Stable; within normal limits; no signs of severe anemia.
	RBC (Million/µL)	4.72 ±0.31	6.58%	p > 0.05	Stable; no significant variance vs. controls.
	Hematocrit (%)	43.0 ±1.9	4.42%	p > 0.05	Highly constant values across the patient cohort.
	WBC (/mm³)	6900 ±1032	15.00%	p > 0.05	Normal range; rules out active leukocytosis.
	Platelets (/mm³)	243,529 ±23,727	9.75%	p > 0.05	Normal and stable baseline values.
Liver function tests (LFT)	Bilirubin Total (mg/dL)	14.8 ±4.8	32.43%	p > 0.05	Normal hepatic clearance with mild, minor fluctuations.
	Bilirubin Direct (mg/dL)	7.7 ±2.8	36.36%	p > 0.05	Normal range; values show minor, insignificant variability.
	Bilirubin Indirect (mg/dL)	7.1 ±2.9	40.85%	p > 0.05	Values display moderate variation but remain nonsignificant.
	AST (U/L)	42.9 ±7.7	17.95%	p > 0.05	Intact hepatic structural integrity.
	ALT (U/L)	117.2 ±8.4	7.17%	p <0.05	Acceptable bounds but shows a minor, statistically detectable variance.
	ALP (U/L)	130.7 ±9.4	7.19%	p > 0.05	Stable; shows no signs of biliary obstruction or bone metastasis activity.
	Total Protein (g/dL)	8.4 ±0.3	3.57%	p > 0.05	Wellpreserved synthetic function.
	Albumin (g/dL)	4.2 ±0.1	2.38%	p > 0.05	Highly stable plasma protein levels.
	Globulin (g/dL)	4.2 ±0.2	4.76%	p > 0.05	Normal range; low overall cohort variability.
	A/G Ratio	1.04 ±0.04	3.85%	p > 0.05	Balanced serum protein distribution.
Renal function tests (RFT)	Urea (mg/dL)	21.6 ±1.7	7.87%	p > 0.05	Completely preserved glomerular function; low variability.
	Creatinine (mg/dL)	1.27 ±0.15	11.81%	p > 0.05	Stable clear renal clearance across the patient population.
	Sodium (Na, mmol/L)	137.0 ±1.0	0.73%	p > 0.05	Tight homeostatic control; lowest overall variation in study.
	Potassium (K, mmol/L)	4.75 ±0.20	4.21%	p > 0.05	Normal electrolyte homeostasis maintained.
Glycemic Control	HbA1c (%)	6.05 ±0.49	8.10%	p > 0.05	Wellcontrolled metabolic blood sugar level; no connection to CRP (p=0.30).

Discussion

This case series aims to highlight the longitudinal patterns of the patients' laboratory profiles, especially the consistency of their hematological, hepatic, and renal parameters. The data gathered from the patient cohort reveal the complex interplay of biological processes, especially the variables' considerable variation. The study's major goal was to measure the patients' PCA3, a prostate cancer-specific RNA, which has been proven to be effective in the detection and monitoring of prostate cancer. The PCA3 expression level ranged from 0.1 to 0.6, averaging about 0.36 and this shows that the majority of the patients had moderate expression. Expression level of PCA3 was slightly positively skewed, suggesting that the majority of patients had moderate expression of the gene, whereas others had higher expression [18].

Alshalalfa and his colleagues (2017) have noted the existence of a bimodal distribution in PCA3 levels in biopsy tissues. Low levels of PCA3 have been significantly correlated with high-grade disease, adverse pathology, metastatic progression, and poor outcomes ($p < 0.001$). However, the sensitivity of PCA3 for high-grade disease (GS≥8) was found to be low at 55%, with 42% of these having low PCA3 [19]. The difference in the expression level of PCA3 is quite consistent with what we know about prostate cancer. Prostate cancer, like all cancer types, is very individualized, meaning the cancer can be quite different from person to person with regards to the severity of the cancer and the way the tumor behaves [20-22]. The Gleason score, a measure of the tumor's differentiation and severity, ranged from 6 to 10 and most patients had a score of about 8, indicating that they had high-grade tumors and our results was similar with the results by Townsend and his colleagues (2013) by meanes of grading system [23]. Wei and his research group (2015) revealed that the high-grade tumors have been correlated to poor patient prognosis [24] and the high Gleason score and PCA3 levels indicate that the majority of the patients in this group had aggressive prostate cancer. This indicates the importance of the integration of biomarkers like PCA3 and traditional grading systems to obtain a more comprehensive patient risk assessment [25-27]. The parameters like hemoglobin, RBC, hematocrit, WBC, and platelet counts for the patients remained within the normal limits and showed no statistically significant variance ($p > 0.05$) when compared against the baseline of the control sample ($n = 21$). The hemoglobin level was between 13.5 and 14.2 g/dL, and the RBC count was about 4.7 million/mL. This indicates that the erythropoietic process was still active, and there was no major problem of anemia. The WBC and platelet counts, i.e., 6000-8000/cumm and 220,000-280,000/mm3, respectively, indicated that there was no major infection, inflammation, or blood disorder. This indicates that, despite the fact that the patients had high-grade tumors, their health status was still maintained, and this is a positive indication for the treatment and prognosis of the patients.The major observation was the fluctuation in the level of CRP, indicating systemic inflammation. Kim and his colleagues (2013) have observed that prostate cancer patient had higher CRP than benign patient [28]. The CRP values ranged from 12 to 45 mg/L, with an average value of about 24.3 mg/L and a high coefficient of variation, about 44%. Notably, these levels represent a significant elevation ($p < 0.05$) relative to the stable baseline provided by the control sample ($n = 21$). The Statistical analysis showed a positive correlation between CRP levels and the Gleason score, indicating that elevated CRP corresponds to a higher tumor grade. This suggests that physiological stress response could be an active participant in the tumor progression, aligning with previous studies that suggest these immune-mediated pathways assist prostate cancer in the advancing, metastasizing and undergoing oncogenic transformation [29-31]. Furthermore, higher CRP values correlated with the increased PCA3 expression, reinforcing idea that this systemic reactivity drives tumor advancement and may accelerate its course. These findings suggest that CRP could serve a dual purpose: as a predictive biomarker for disease progression, and as a therapeutic target where modulating this systemic response could effectively slow tumor growth [32].

All the liver function parameters of the cohort, such as bilirubin (total, direct, and indirect) as well as AST, ALT, and ALP, were generally within acceptable physiological bounds. Compared to the control sample ($n = 21$), a minor but statistically detectable variance was noted in ALT ($p <0.05$); however, the overall profile did not indicate any liver dysfunction or metastasis as observed in prostate cancer patients. The constant values of the liver enzymes indicate that the systemic effect of the prostate cancer did not affect the liver functions, which is crucial when administering chemotherapy or hormone therapy as part of the treatment regimens [33, 34]. The kidney function tests such as urea and creatinine also had constant values, averaging 21.6 mg/dL and 1.27 mg/dL, respectively, mirroring the stable renal clearance seen in the control sample ($n = 21$) with no significant difference ($p > 0.05$). The balance of the body's electrolytes, such as sodium and potassium, also had constant values within the normal range, showing good kidney function [35], with sodium demonstrating the tightest homeostatic control across the study population. Since there is no sign of kidney dysfunction, it shows that the progress of prostate cancer did not affect the kidneys as seen in patients with paraneoplastic syndromes, which is due to the effects of cancer on the body or the metastasis of cancer cells. The glycemic control levels, as reflected by HbA1c levels, were between 5.5 and 6.4 percent, showing well-controlled blood sugar levels among diabetic patients, which did not deviate significantly from the control sample ($n = 21$) values ($p > 0.05$). This indicates that the metabolic dysfunction, such as uncontrolled diabetes, did not influence progression of the disease. Currently, relationship between metabolic syndrome, prostate cancer, and systemic immune activation is being actively investigated with evidence suggesting that metabolic disruptions can significantly modulate malignancy [36-38]. Such conditions need to be monitored to understand how cancer is related to metabolic

problems and how it can be treated holistically.

Conclusion

The case series demonstrates the overall physiological stability of hematological, hepatic, and renal functions in prostate cancer patients over time, which remained remarkably consistent when compared against the healthy baseline of the control sample (n = 21), despite notable changes in molecular and inflammatory markers. Interestingly, PCA3 levels were found to vary between 0.1 and 0.6. Higher levels of PCA3 were found to be related to higher Gleason scores; hence, indicating a more aggressive disease state. Hematological parameters were found to be within normal physiological limits and did not deviate significantly from the control sample (n = 21), which shows the systemic health of the patients was well maintained. However, the results for the CRP protein showed considerable variation and a statistically significant elevation compared to the control sample (n = 21), while indicating a positive correlation with both Gleason scores and PCA3 protein levels. Furthermore, liver and kidney function tests were found to be normal and stable relative to the control group baseline, showing that no significant systemic organ dysfunction existed. This preserved organ function would help in the easier management and therapeutic tolerability of the patients. By using the advanced cluster analysis method, different types of prostate cancer patients were identified. This underscores the potential benefits that can be derived from an integrated approach to patient management, though regular longitudinal observation is necessary to obtain the maximum clinical benefits.

Consent for publication

All participants gave their informed consent before participation from the Department of Urology, AIIMS Bhopal after fulfilling the inclusion and exclusion criteria and after obtaining consent.

Declaration of conflict of interests

No.

CRediT authorship contribution statement

DA, AK, ASG and DK collected the data. SKR and SM conceptualized the manuscript. SKR wrote the first draft. SR, ShM and SM edited it. All the authors agreed and approved the final manuscript.

Author's Disclosures

The authors declare no conflicts of interest related to this work. Financial support was received from AIIMS Bhopal, India, Intramural fund (Reference Number: IHEC/SR/2024/111 Date: 01/02/2024). All authors have approved the manuscript and agree to be accountable for all aspects of the work.

Funding

AIIMS Bhopal, India, Intramural fund (AIIMSBPL/IHECSR/Nov-2024/29 dated: 23rd Dec 2024) of AIIMS Bhopal, India.

Acknowledgments

None.

References

1. Schafer EJ, Laversanne M, Sung H, Soerjomataram I, Briganti A, Dahut W, Bray F, Jemal A. Recent patterns and trends in global prostate cancer incidence and mortality: an update. Eur Urol. 2025;87(3):302–313.
2. Vlajnic T, Bubendorf L. Molecular pathology of prostate cancer: a practical approach. Pathology. 2021;53(1):36–43.
3. Huang Y, Mao J, Li X. Emerging biomarkers in prostate cancer diagnosis and treatment: insights into genetic, RNA and metabolic markers. Int J Oncol. 2026;68(2):15.
4. Lemos AEG, Matos ADR, Ferreira LB, Gimba ERP. The long non-coding RNA PCA3: an update of its functions and clinical applications as a biomarker in prostate cancer. Oncotarget. 2019;10(61):6589–6603.
5. Qin Z, Yao J, Xu L, Xu Z, Ge Y, Zhou L, Zhao F, Jia R. Diagnostic accuracy of PCA3 level in patients with prostate cancer: a systematic review and meta-analysis. Int Braz J Urol. 2020;46(5):691–704.
6. Agosti V, Munari E. Histopathological evaluation and grading for prostate cancer: current issues and crucial aspects. Asian J Androl. 2024;26(6):575–581.
7. Huang J, Reilly KH, Zhang HZ, Wang HB. Clinical evaluation of prostate cancer gene 3 score in diagnosis among Chinese men with prostate cancer and benign prostatic hyperplasia. BMC Urol. 2015;15:118.
8. Watts EL, Perez-Cornago A, Kothari J, Allen NE, Travis RC, Key TJ. Hematologic markers and prostate cancer risk: a prospective analysis in UK Biobank. Cancer Epidemiol Biomarkers Prev. 2020;29(8):1615–1626.
9. Zhou K, Li C, Chen T, Zhang X, Ma B. C-reactive protein levels could be a prognostic predictor of prostate cancer: a meta-analysis. Front Endocrinol (Lausanne). 2023;14:1111277.
10. Jensen GL, Naziri J, Hammonds KP, Jhavar SG, Swanson G. C-reactive protein is a poor marker of baseline inflammation in prostate cancer and response to radiotherapy or androgen ablation. Cureus. 2021;13(11):e19639.Stark T, Livas L, Kyprianou N. Inflammation in prostate cancer progression and therapeutic targeting. Transl Androl Urol. 2015;4(4):455-63. doi: 10.3978/j.issn.2223-4683.2015.04.12. PMID: 26816843; PMCID: PMC4708587
11. Karzai FH, Madan RA, Dahut WL. Metabolic syndrome in prostate cancer: impact on risk and outcomes. Future Oncol. 2016;12(16):1947–1955.

12. Yanase T, Kawanami T, Tanaka T, Tanabe M, Nomiyama T. Impact of metabolic disorders on prostate cancer growth: androgen and insulin resistance perspectives. Reprod Med Biol. 2017;16(3):252–257.
13. Loeb S, Curnyn C, Sedlander E. Perspectives of prostate cancer patients on Gleason scores and the new grade groups: initial qualitative study. Eur Urol. 2016;70(6):1083–1085.
14. Hamed NW, Elbeljihy HS, Hussin SA, Fouda RM, Oy EK, Magar RW. From prostate specific antigen to genomic signatures: advances in biomarkers for prostate cancer diagnosis and prognosis. Transl Oncol. 2026;66:102719.
15. Majewska Z, Zajkowska M, Pączek S, Nowiński AR, Sokólska W, Gryko M, Orywal K. The clinical relevance of tumor biomarkers in prostate cancer: a review. Cancers (Basel). 2025;17(23):3742.
16. Fernandes R, Costa C, Fernandes R, Barros AN. Inflammation in prostate cancer: exploring the promising role of phenolic compounds as an innovative therapeutic approach. Biomedicines. 2023;11(12):3140.
17. Marks LS, Bostwick DG. Prostate cancer specificity of PCA3 gene testing: examples from clinical practice. Rev Urol. 2008;10(3):175–181.
18. Alshalalfa M, Verhaegh GW, Gibb EA, Santiago-Jiménez M, Erho N, Jordan J, Yousefi K, Lam LLC, Kolisnik T, Chelissery J, Seiler R, Ross AE, Karnes RJ, Schaeffer EM, Lotan TT, Den RB, Freedland SJ, Davicioni E, Klein EA, Schalken JA. Low PCA3 expression is a marker of poor differentiation in localized prostate tumors: exploratory analysis from 12,076 patients. Oncotarget. 2017;8(31):50804–50813.
19. Malik A, Srinivasan S, Batra J. A new era of prostate cancer precision medicine. Front Oncol. 2019;9:1263.
20. Hamid AA, Sweeney CJ, Hovens C, Corcoran N, Azad AA. Precision medicine for prostate cancer: an international perspective. Urol Oncol. 2024;42(12):392–401.
21. Mytsyk Y, Borzhiyevs'kyy A, Kobilnyk Y, Shulyak AV, Dutka I, Borzhiyevs'kyy O, Górecki A. Personalized management of prostate cancer: from molecular and imaging markers to radiogenomics. Pol J Radiol. 2022;87:e58–e62.
22. Townsend NC, Ruth K, Al-Saleem T, Horwitz EM, Sobczak M, Uzzo RG, Viterbo R, Buyyounouski MK. Gleason scoring at a comprehensive cancer center: what's the difference? J Natl Compr Canc Netw. 2013;11(7):812–819.
23. Wei W, Leng J, Shao H, Wang W. High PCA3 scores in urine correlate with poor prognostic factors in prostate cancer patients. Int J Clin Exp Med. 2015;8(9):16606–16612.
24. Davalan W, Alkins R. Prognostic and predictive determinants in high-grade gliomas: integrating tumor-intrinsic biology with patient and system-level factors. Front Neurol. 2025;16:1664458.
25. Crocetto F, Musone M, Chianese S, Conforti P, Digitale Selvaggio G, Caputo VF, Falabella R, Del Giudice F, Giulioni C, Cafarelli A, Lucarelli G, Busetto GM, Ferro M, Barone B, Zattoni F, Terracciano D. Blood and urine-based biomarkers in prostate cancer: current advances, clinical applications, and future directions. J Liq Biopsy. 2025;9:100305.
26. Guo Y, Chen W, Zhu J, Wang X, Ding Y, Duan L, Mai Y, Bai Y, Niu Y, Zhao Y. Prostate cancer associated 3 (PCA3) is a potential indicator of pathological grades of prostate cancer. Discov Oncol. 2026;[Epub ahead of print].
27. Kim Y, Jeon Y, Lee H, Lee D, Shim B. The prostate cancer patient had higher C-reactive protein than BPH patient. Korean J Urol. 2013;54(2):85–88.
28. Zhou M, Liang J, Hui J, Xu J. Inflammation-related indicators have potential to improve overall quality of prostate cancer management: a narrative review. Transl Androl Urol. 2023;12(5):809–822.
29. Chen X, Li Y, Li G, Zhang X, Xie G, Huang Y, Yin H. Clinical significance of serum high sensitive C-reactive protein/albumin ratio in primary prostate biopsy. Front Oncol. 2024;14:1325524.
30. Sciarra A, Gentilucci A, Salciccia S, Pierella F, Del Bianco F, Gentile V, Silvestri I, Cattarino S. Prognostic value of inflammation in prostate cancer progression and response to therapy: a critical review. J Inflamm (Lond). 2016;13:35.
31. Du J, Lan J, Xiong J, Yang H, Xu X, Tang C, Huang G, Ying Q, Mu J, Hu Q. Efficiency of C-reactive protein in prognosis evaluation of prostate cancer: a systematic review and meta-analysis. Transl Cancer Res. 2021;10(10):4432–4439.
32. Archer M, Dogra N, Kyprianou N. Inflammation as a driver of prostate cancer metastasis and therapeutic resistance. Cancers (Basel). 2020;12(10):2984.
33. Ali S, Zehra A, Khalid MU, Hassan M, Shah SIA. Role of C-reactive protein in disease progression, diagnosis and management. Discoveries (Craiova). 2023;11(4):e179.
34. Ghoshal A, Garmo H, Hammar N, Jungner I, Malmström H, Walldius G, Van Hemelrijck M. Can pre-diagnostic serum levels of sodium and potassium predict prostate cancer survival? BMC Cancer. 2018;18(1):1169.
35. Rhee H, Vela I, Chung E. Metabolic syndrome and prostate cancer: a review of complex interplay amongst endocrine factors in pathophysiology and progression. Horm Cancer. 2016;7(2):75–83.
36. Monroy-Iglesias MJ, Russell B, Crawley D, Allen NE, Travis RC, Perez-Cornago A, Van Hemelrijck M, Beckmann K. Metabolic syndrome biomarkers and prostate cancer risk in the UK Biobank. Int J Cancer. 2021;148(4):825–834.
37. Guerrios-Rivera L, Howard LE, Wiggins EK, Hoyo C, Grant DJ, Erickson TR, Ithisuphalap J, Freedland AR, Vidal AC, Fowke JH, Freedland SJ. Metabolic syndrome is associated with aggressive prostate cancer regardless of race. Cancer Causes Control. 2023;34(3):213–221.

Copyright© 1999–2026 International Federation of Clinical Chemistry and Laboratory Medicine (IFCC). All rights reserved.
This is a Platinum Open Access Journal distributed under the terms of the Creative Commons Attribution Non-Commercial License, which permits unrestricted non-commercial use, distribution, and reproduction in any medium, provided the original work is properly cited.