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CLINICAL SIGNIFICANCE OF SERUM PROSTATE SPECIFIC ANTIGEN IN PROSTATE CANCER PATIENTS AND ITS RELATIONSHIP WITH HYPERCHOLESTEROLEMIA

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ABSTRACT

Introduction: *Prostate cancer, prevalent among men, necessitates early screening, especially with the increasing Westernization of lifestyles, including high-fat diets, which have raised its incidence. Hypercholesterolemia has been linked to aggressive prostate cancer, emphasizing the importance of dietary factors. This study examines the correlation between PSA levels and lipid profiles, specifically total cholesterol, low-density lipoprotein (LDL), high-density lipoprotein (HDL), and triglycerides, in prostate cancer patients.*

Methods: *Fifty male patients (60-89 years) with biopsy-confirmed prostate carcinoma were included. Fasting blood samples were analysed for PSA, total cholesterol (TC), triglycerides (TG), high-density lipoprotein (HDL), and low-density lipoprotein (LDL). PSA was quantified using immunoassay, while lipid parameters were measured via enzymatic colorimetric methods. The statistical analysis was performed via ANOVA and Pearson's correlation.*

Results: *All patients with histopathologically confirmed prostate cancer exhibited elevated PSA levels, confirming 100% diagnostic sensitivity. PSA demonstrated a significant positive correlation with total cholesterol ($r = 0.79$) and LDL ($r = 0.59$), and an inverse correlation with HDL ($r = -0.40$) and triglycerides ($r = -0.54$). Group-wise analysis showed higher PSA and lipid abnormalities in patients aged 70–79 years.*

Conclusion*: The data indicate that men with higher PSA levels are more likely to have elevated cholesterol and LDL. Besides, PSA levels were inversely related to triglycerides and HDL, suggesting that patients with high PSA levels might be at increased risk for cardiovascular diseases rather than prostate cancer alone.*

Keywords: *Fasting lipid profile; Hypercholesterolemia; Prostate tissue biopsy; Prostate cancer; Serum prostate specific antigen*

1. INTRODUCTION

Cancer, a disease characterized by pathologic modifications to the inherent process of cell division, is now regarded as major health issue that kills a great deal of individuals each year all over the world. Over 19.3 million cases (or 19,300,000 new ones) of cancer were identified and noted recently; according to the cited numbers, this will result in over 10 million fatalities in 2020. [1]. A number of gene modifications may have a role in the origin of cancer, as shown by the several stages of cancer that researchers have uncovered. These gene changes lead to abnormal cell growth. The existence of genetic illnesses caused by inheritance or heredity is a major factor in the increase in cell proliferation. The highest percentages of cancer types in men are found in the prostate, lung and bronchus, colon and rectum, and bladder, correspondingly [2].

Prostate cancer is one of the most well-known illnesses that affect men. It is the kind of cancer that is most common diagnosed in men in America [3]. On prostate gland periphery 70% adenocarcinoma are found, and the precancerous lesion that most adenocarcinomas exhibit is intraepithelial neoplasia. A sequence of initiating and promotional events under the influence of heredity and environment give rise to malignant change in the prostate cells and subsequent progression to cancer. Major risk factors that influence the course of the disease include dietary variables, high cholesterol intake, environmental carcinogens, hormonal milieu, racial and geographic disparities, and ethnicity [4]. Although it is not fully understood, prostate cancer shares a hereditary component with other cancers. If there is a family history of the illness, men are more likely to get it [5]. The signs of prostate cancer may take years to manifest, and the cancer can grow very slowly. The symptoms include feeling as though you can't completely empty your bladder, passing pee more frequently than normal, especially at night, and needing to rush to the bathroom. Unusual symptoms include pain when urinating and blood in the pee or semen [3]. Early prostate cancer screening is more important now than ever since westernization of lifestyle, particularly the adoption of high-fat diets, has raised the prevalence of prostate cancer in nations where the risk was lowest before. Indeed, hypercholesterolemia has also been connected to aggressive prostate cancer, and a number of diets have been linked to advanced prostate cancer [6]. To this day, the accepted biochemical prostate cancer screening test is serum prostate specific antigen (PSA) [7], it is because PSA is only secreted by prostate epithelial cells [8]. This enzyme acts as a liquifactant of the seminal coagulum subsequent to its high concentration secretion into the prostatic ductal system. Early research by Myrtle and Ivor (1989) defined normal PSA levels as falling between 0 and 4.0ng/mL [9]. Age wise normal range for PSA as following for all ages it is <4.0 ng/mL, for 40-49 it is <2.5 ng/mL, for 50-59 it is <3.5 ng/mL, for 60-69 it is <4.5 ng/mL and for greater than 70 years it is <6.5 ng/mL [10]. Prostatic carcinoma clinical progression and the likelihood of surgical therapy connected to prostatic carcinoma can be predicted by PSA. Prostate volumes and PSA are typically higher in patients who have more severe symptoms associated with prostate cancer. Therefore, PSA is an excellent prognostic and screening tool. When utilized properly, it can result in a prostate cancer early diagnosis [10]. The main purpose of this research work is to evaluate serum PSA diagnostic efficacy in detecting prostate cancer in comparison to tissue biopsy. Moreover, this study also investigates the correlation between hypercholesterolemia and the prognosis of cancerous prostate in prostatic carcinoma patients who have been diagnosed.

2. Materials and methods

2.1. Selection of study cohort

Patients with prostate cancer, between the ages of 60 and 90, who visited the oncology department for routine therapy and follow-ups were selected. The diagnosis of prostate cancer in these patients was made using the tumor biomarker serum PSA test, and confirmed by prostate gland tissue biopsy. According to world health organization (WHO) sample size calculator, sample size for this study was kept fifty i.e. fifty prostate cancer patients were selected to assess the significance of PSA and its relationship with

hypercholesterolemia. Patients suffering from any other cancer were excluded from study. Patients with age lesser then sixty and greater than ninety were also excluded from study.

Personal data of patients i.e., name, address and phone number were kept confidential. The analysis was performed according to ethical guidelines after obtaining ethical approval (approval number: 425) from institutional review board of Combined Military Hospital (CMH) Rawalpindi and informed consents signed by patients.

2.2. Blood sampling

From identified prostate cancer patients, blood was collected in early morning fasting condition, in yellow top vials and then centrifuged at 500 g for 15 minutes to collect the serum for biochemical analysis.

2.3. Assessment of lipid profile

2.3.1. Total cholesterol estimation

Kit method was used for the estimation of cholesterol. The reaction mixture was prepared by adding reagent to the test sample (19 µL). The reagent consists of cholesterol esterase, cholesterol oxidase, 4-aminoantipyrine (4-AAP) and peroxidase in phosphate buffer. Afterwards, it was incubated for 5 minutes at 37 °C followed by the measurement of absorbance at 460 nm by Cobas c 303 chemistry analyzer. The concentration of total cholesterol in each sample was displayed for analysis [3,11].

2.3.2. Triglyceride estimation

Kit method was used for the estimation of triglyceride. The reaction mixture was prepared by adding reagent to the test sample (19 µL). The reagent consists of glycerol kinase, glycerol-3-phosphate oxidase and peroxidase in Tris buffer. Afterwards, it was incubated for 5 minutes at 37 °C followed by the measurement of absorbance at 460 nm by Cobas c303 chemistry analyzer. The concentration of triglyceride in each sample was displayed for analysis [3].

2.3.3. High-density lipoprotein estimation

Kit method was used for the estimation of HDL. The reaction mixture was prepared by adding reagent to the test sample (19 µL). The reagent consists of cholesterol esterase, cholesterol oxidase, 4-aminoantipyrine (4-AAP) and peroxidase in phosphate buffer. Afterwards, it was incubated for 5 minutes at 37 °C followed by the measurement of absorbance at 460 nm by Cobas c303 chemistry analyser. The concentration of HDL in each sample was displayed for analysis [3].

2.3.4. Low-density lipoprotein estimation

LDL cholesterol is calculated by using Fried Ewald formula LDL= TC – (HDL – TG/5 (measured in mg/dL)) [3,12].

2.4. Statistical methods

Statistical package for social sciences no. 22 was used for analysis of this study data. ANOVA revealed the mean of data according to age grouping. Pearson correlation was use to evaluate the relationship between deranged PSA and hypercholesterolemia. Graphical analysis of data was done by using MS Excel [13,14].

3. Results

3.1. Comparison of biopsy report with prostate specific antigen results

Prostate-specific antigen (PSA) remains one of the most widely utilized and clinically validated tumor biomarkers for the detection and management of prostate cancer. In the present study, all 50 patients with biopsy-confirmed prostate carcinoma exhibited elevated serum PSA levels (> 4 ng/mL). Conversely, individuals with normal PSA concentrations showed no histological evidence of malignancy. It reflects a 100% sensitivity of PSA in identifying prostate cancer within this patient sample. Therefore, the result interpreted a direct and positive correlation between elevated PSA levels and the presence of prostate malignancy making it an essential biomarker not only in the initial screening and risk stratification of prostate cancer but also in the prognosis and cancer recurrence assessment of patients. A graphical representation comparing PSA levels with corresponding biopsy results is provided in Fig. 1.

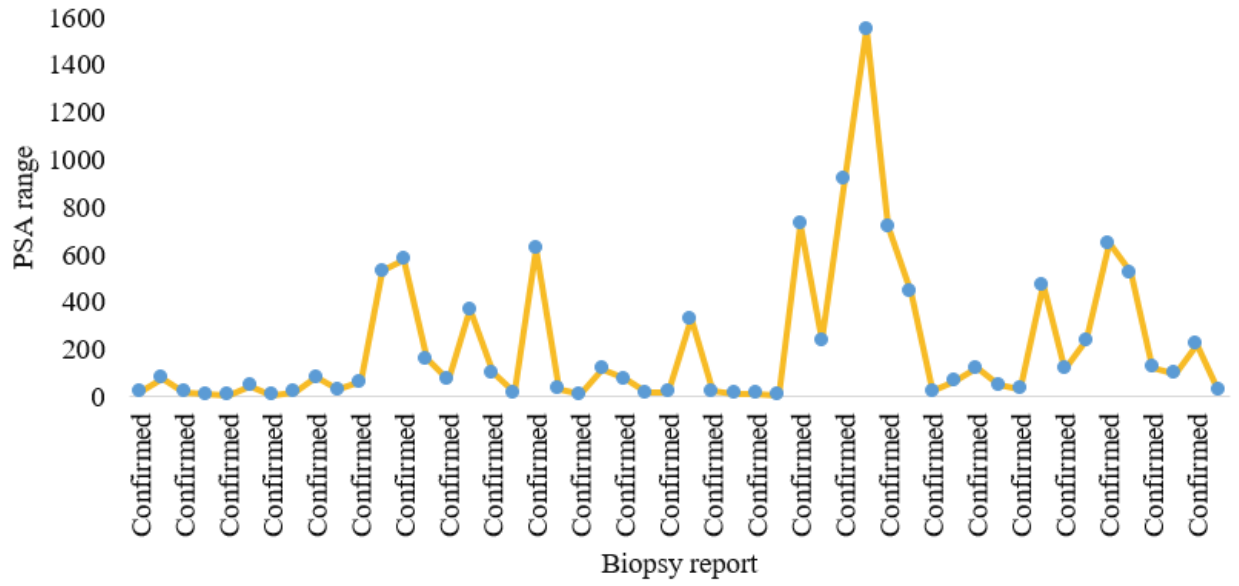

Fig. 1. Serum prostate specific antigen and biopsy comparison

3.2. Relationship of prostate specific antigen and low density lipoprotein

An evident positive correlation between PSA levels and low-density lipoprotein (LDL) cholesterol concentrations was identified, as demonstrated in Fig. 2. Several biological mechanisms may explain this association. One proposed explanation is that cholesterol, primarily carried by LDL particles, may enhance prostate cell proliferation by altering membrane lipid composition and influencing androgen synthesis, thereby promoting tumorigenesis [15]. Another possible mechanism is that increased LDL cholesterol may contribute to subclinical inflammation within the prostate gland, leading to raised PSA levels [16]. However, it is essential to interpret this correlation with caution as elevated LDL cholesterol alone should not be considered an indicator of prostate cancer. Instead, it suggests a potential risk association, indicating that men with high LDL cholesterol may have an increased predisposition to prostate-related abnormalities. Accordingly, maintaining LDL cholesterol within physiologically acceptable limits may support overall prostate health. This can be achieved through dietary regulation, routine physical activity, and pharmacological intervention.

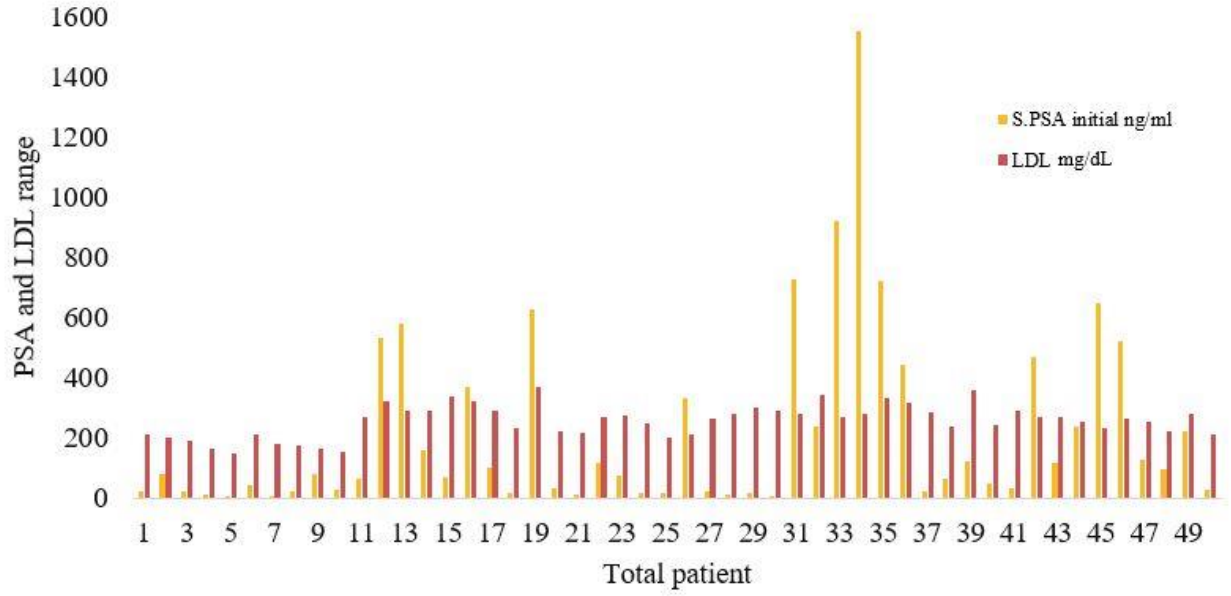

Fig. 2. Relationship between prostate specific antigen and low-density lipoprotein.

3.3. Relationship of prostate specific antigen and total cholesterol

A positive correlation was observed between serum prostate-specific antigen (PSA) levels and total cholesterol concentrations, as illustrated in Fig. 3. This trend suggests that an increase in total cholesterol is generally associated with elevated circulating PSA levels. Prior studies have also reported that hypercholesterolemia is associated with an increased risk of prostate cancer and elevated PSA concentrations in men [17]. However, it is noteworthy that two out of the fifty participants in this study did not follow this trend. Several factors may account for this deviation. These individuals may possess other dominant risk factors for prostate cancer, such as advanced age, genetic predisposition, or ethnic background that obscure the influence of cholesterol. Additionally, the use of medications, including statins or androgen-depleting agents, could modulate PSA levels independently of cholesterol status [18]. Besides, the graphical data suggest a strong positive association between serum total cholesterol and PSA levels, supporting the hypothesis of a metabolic influence on prostate health.

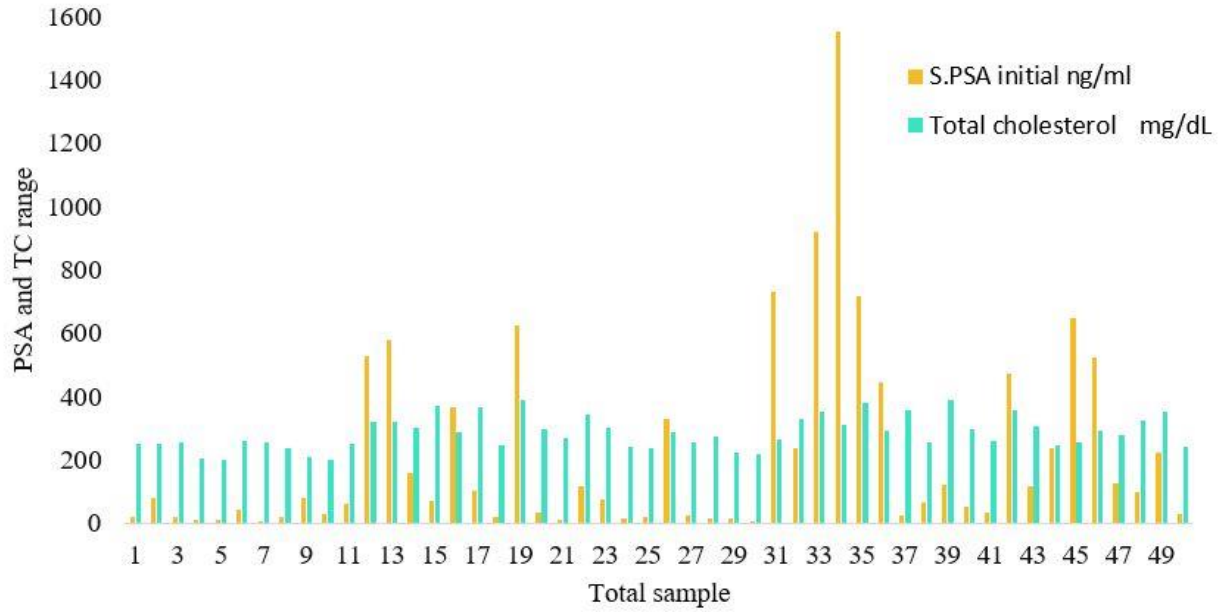

Fig. 3. Relationship between prostate specific antigen and total cholesterol.

3.4. Relationship of prostate specific antigen and high-density lipoprotein

A complex inverse correlation was observed between serum PSA and high density lipoprotein (HDL), as illustrated in Fig. 4. This relationship may be attributed to their shared involvement in inflammatory processes. Inflammatory conditions are known to downregulate hepatic HDL synthesis while concurrently upregulating PSA production [19]. An alternative mechanism involves a direct molecular interaction between PSA and HDL. Previous studies have indicated that PSA may bind to HDL, thereby impairing their capacity to facilitate reverse cholesterol transport. Such interference could contribute to diminished HDL levels in patients with elevated PSA concentrations [19,20]. Furthermore, variables such as age and circulating testosterone levels may influence this association. Testosterone, which declines with age, plays a regulatory role in modulating both PSA secretion and HDL metabolism [21]. This hormonal link might help explain the stronger inverse relationship noted between PSA and HDL in older men. Considering HDL established cardioprotective role, elevated PSA levels may serve not only as a marker for prostate pathology but also as a potential indicator of increased cardiovascular risk.

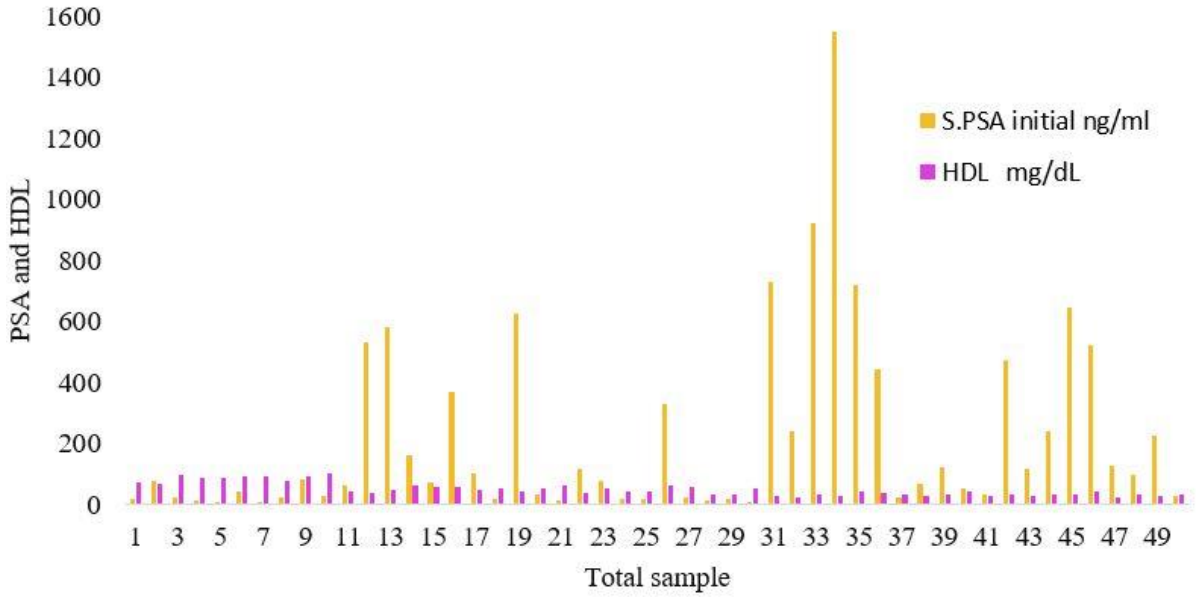

Fig. 4. Relationship between prostate specific antigen and high-density lipoprotein.

3.5. Relationship of prostate specific antigen and triglyceride

Fig. 5 illustrates a statistically significant inverse correlation between serum triglyceride levels and PSA concentrations. In all 50 patients included in this study, triglyceride levels were either within normal limits or low, while PSA levels were elevated. However, variability among individual data points suggests that the association is not perfectly linear while the pattern of inverse relation remains consistent. This pattern holds clinical significance, as elevated triglyceride levels may obscure PSA based detection of early stage prostate cancer. In men with hypertriglyceridemia, reduced PSA concentrations may potentially lead to under diagnosis or delayed detection of malignancy. The age group-wise mean ± standard error of mean (SEM) values for prostate cancer patients are summarized in Table 1.

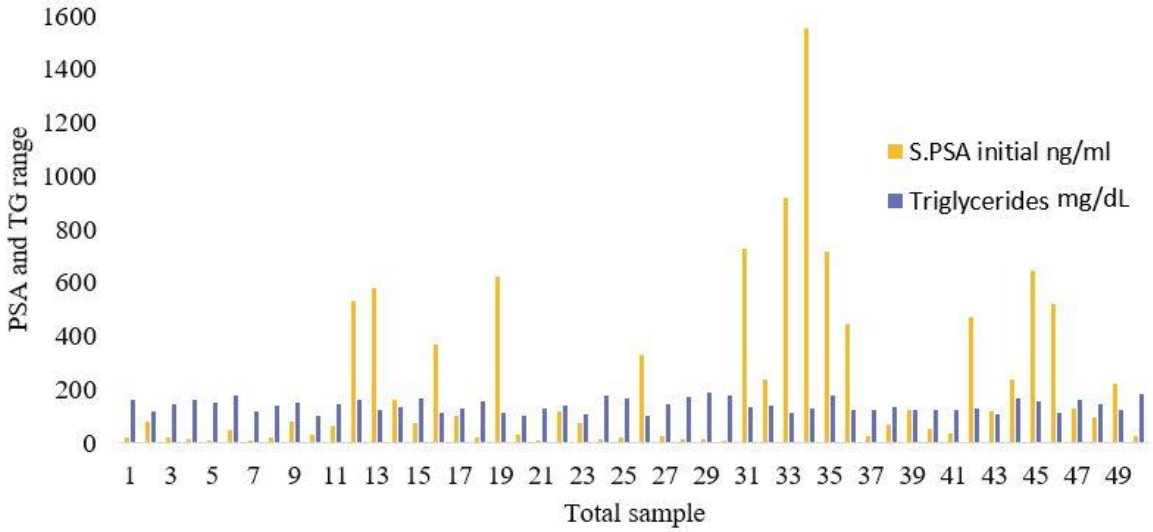

Fig. 5. Relationship between prostate specific antigen and triglycerides.

Table 1
Mean ± Standard Error of Mean of age group data of prostate cancer patients.

Group	Age (Yrs.)	Mean ± SEM				
		PSA (ng/mL)	TC (mg/dL)	TG (mg/dL)	HDL (mg/dL)	LDL (mg/dL)
G-I	60 to 69	68.0 ± 5.07	262.9 ± 1.7	134.5 ± 3.52	62.3 ± 1.36	217.6 ± 3.59
G-II	70 to 79	150.3 ± 8.91	290.8 ± 1.28	138.3 ± 7.96	45.7 ± 1.51	266.5 ± 3.30
G-III	80 to 89	120.1 ± 7.0	288.2 ± 1.90	146.1 ± 7.96	45.0 ± 8.71	262.7 ± 2.85

The ANOVA revealed that the overall means for the parameters, PSA, cholesterol, LDL, HDL, and triglycerides within all age ranges were significantly ($P<0.05$) different from each other. However, repeated measures ANOVA (Dunnett's T3 Post Hoc Test) between various age groups revealed that the PSA in G-1 patients was significantly lower than the G-II and G-III groups. The data presented in Table 2 shows the mean plus standard error mean of different age groups. The results revealed that PSA level in G-II patients was increased significantly (150.3) as compared to G-I patients (68.0) and G-III patients (120.1) and overall increased than the normal PSA levels.

Table 2
The Mean ± SEM of the different age groups of prostate cancer patients.

Groups				
Parameters (Normal range and units)	Mean ± SEM			
	G-I	G-II	G-III	Overall
PSA (4.0 ng/mL)	68.0 ± 5.07^{a}	150.3 ± 8.91^{b}	120.1 ± 7.0^{c}	112.8 ± 6.9
Cholesterol (<200 mg/dL)	262.7 ± 1.70^{a}	290.8 ± 1.40^{b}	288.2 ± 1.82^{c}	280.5 ± 1.64
Triglyceride (<150 mg/dL)	134.5 ± 1.42^{a}	138.3 ± 8.38^{b}	146.1 ± 9.97^{c}	139.6 ± 6.59
HDL (>60 mg/dL)	62.2 ± 1.37^{a}	45.5 ± 1.45^{b}	45.9 ± 1.60^{c}	51.2 ± 1.42
LDL (<130 mg/dL)	217.4 ± 3.56^{a}	266.6 ± 3.30^{b}	262.7 ± 2.65^{bc}	248.9 ± 3.17

Different superscript (a, b, c) shows the statistical difference ($P<0.05$) between the groups.

Blood cholesterol was raised significantly in G-II patients (290.8) as compared to G-I patients (262.7) and G-III patients (288.2) and overall increased than the normal blood cholesterol level. Serum triglyceride was raised significantly in G-III patients (146.1) as compared to G-I patients

(134.5) and G-II patients (138.3) but overall decreased than the normal triglycerides level. Similarly, data revealed that Serum HDL was elevated significantly in G-I patients (62.2) as compared to G-II patients (45.5) and G-III patients (45.9) but overall decreased than the normal HDL level. Serum LDL was raised significantly in G-II patients (266.6) as compared to G-I patients (217.4) and G-III patients (262.7) but overall increased than the normal LDL levels.

3.6. Correlation of prostate specific antigen with lipid profile in group-I patients

The result of correlation coefficient of PSA with cholesterol, triglycerides, HDL and LDL of G-I group patients is given in Table 3. PSA × cholesterol (r=0.79; P<0.01) and PSA × LDL (r=0. 0.17; P<0.05) had a statistical significantly positive correlation. PSA × triglycerides (r = -0.54; P<0.01) and PSA × HDL (r= -0.08; P<0.05) had a statistical significantly negative correlation.

Table 3
Pearson's correlation coefficient (r) of PSA in G-I group patients.

Parameters	G-I group patients
PSA × Cholesterol	0.79 **
PSA × Triglycerides	-0.54**
PSA × HDL	-0.08*
PSA × LDL	0.17*

**Correlation (r) is significant at the *P* 0.01 level (2-tailed), *Correlation is significant at the *P* 0.05 level (2-tailed) and NS for non-significant.

3.7. Correlation of prostate specific antigen with lipid profile in group II patients

The result of correlation coefficient of PSA with cholesterol, triglycerides, HDL and LDL of G-II group patients is given in Table 4. PSA × cholesterol (r=0.36; P<0.01) and PSA × LDL (r=0.28; P<0.01) had a statistical significantly positive correlation. PSA × triglycerides (r = -0.34; P<0.01) and PSA × HDL (r= -0.40; P<0.01) had a statistical significantly negative correlation.

Table 4
Pearson's correlation coefficient (r) of PSA in G-I1 group patients.

Parameters	G-II group patients
PSA × Cholesterol	0.36 **
PSA × Triglycerides	-0.34**
PSA × HDL	-0.40**
PSA × LDL	0.28**

**Correlation (r) is significant at the *P* 0.01 level (2-tailed), *Correlation is significant at the *P* 0.05 level (2-tailed) and NS for non-significant.

3.8. Correlation of prostate specific antigen with lipid profile in group-III patients

The result of correlation coefficient of PSA with cholesterol, triglycerides, HDL and LDL of G-III group patients is given in Table 5. PSA × cholesterol (r=0.65; P<0.01) and PSA × LDL (r=0.59; P<0.01) had a statistical significantly positive correlation. PSA × triglycerides (r = -0.17; P<0.05) and PSA × HDL (r= -0.31; P<0.01) had a statistical significantly negative correlation.

Table 5
Pearson's correlation coefficient (r) of PSA in G-I1I group patients.

Parameters	G-III group patients
PSA × Cholesterol	0.65**
PSA × Triglycerides	-0.17*
PSA × HDL	-0.31**
PSA × LDL	0.59**

**Correlation (r) is significant at the *P* 0.01 level (2-tailed), *Correlation is significant at the *P* 0.05 level (2-tailed) and NS for non-significant.

4. Discussion

Serum PSA serves as a vital indicator for examining and diagnosing prostate cancer, showcasing a high relationship among its levels and the presence of the disease [22]. The prostate gland produces a protein called PSA, and its concentration in the blood can be measured through a blood test. In the context of prostate cancer, elevated PSA levels are often indicative of abnormal cellular activity within the prostate [23]. PSA assay is the most efficacious cancer screening test for prostate cancer (PCa). A study conducted in Nigeria found 96% sensitivity [24]. In this study, all the cases reported positive PSA test and have prostate cancer when confirmed by prostate biopsy. Strong correlation between PSA levels and prostate cancer, highlighting PSA as a premier tumor biomarker for diagnosis. This recognition establishes PSA as a valuable diagnostic tool, facilitating the early detection and monitoring of prostate cancer, thereby aiding in clinical management and treatment decisions.

Since serum PSA has a shorter reporting period (24 hours) than biopsy, which is more costly and takes longer to report (7-10 days) it is more cost-effective [25]. Therefore, we can save money and patient time by employing PSA for screening. Additionally, the patient's therapy can begin right away.

Prostate cancer and dyslipidemia or hypercholesterolemia are significantly correlated [26] and the intended research found similar strong correlation between dyslipidemia or abnormal lipid profile and prostate cancer. The first evidence that cholesterol contributes to prostate disease dates back to Swyer's 1942 study, which found that prostatic adenomas had greater cholesterol levels than normal tissue [27]. The assertion that cholesterol contributes to prostate disease is rooted in scientific observations and research findings that have uncovered links between elevated cholesterol levels and prostate health [16]. Research studies have identified associations between high cholesterol levels and an increased risk of prostate diseases. Cholesterol imbalances may contribute to inflammation, oxidative stress, and alterations in cellular signaling pathways within the prostate gland [28-30].

This research adds credence to previous recent studies indicating a possible increased risk of prostate cancer in white men with hypercholesterolemia. Low HDL was found to be strongly related with prostate cancer, while high LDL was found to be significantly connected with an increased risk of prostate cancer [31]. The results of the previous study, which show a connection between high total cholesterol and prostate cancer, are also supported by our research. Additionally, our research shows that there is a direct correlation between LDL and HDL and an inverse link with prostate cancer. Levels of LDL cholesterol and total cholesterol increase, the risk or incidence of prostate cancer also increases. The correlation between LDL and total cholesterol with prostate cancer suggests a potential role of cholesterol in the development or progression of prostate cancer [32,33]. It may imply that high cholesterol levels could be a risk factor for prostate cancer, although the exact mechanisms linking cholesterol to prostate cancer may vary and may involve complex biological pathways. In the near run, hypercholesterolemia may raise the risk of prostate cancer overall. It is probable that prostate cancer is not the cause of

hypercholesterolemia, but rather one of its effects [34,35].

5. Conclusions

Serum PSA, is a vital biomarker for identifying and tracking prostate cancer. All patients with biopsy-proven malignancy exhibited raised PSA levels, supporting its role as an essential tool in early diagnosis. Additionally, a notable association was observed between PSA and lipid abnormalities, particularly elevated total cholesterol and LDL levels, while inverse relationships were found with HDL and triglycerides. The results interpreted that dyslipidemia, especially hypercholesterolemia, may be involved in the biological mechanisms leading to the progression of prostate cancer. Therefore, monitoring lipid profiles along with PSA may improve risk assessment and preventive strategies. Besides, further research is still required to clarify the role of lipid metabolism in prostate cancer development and to evaluate whether controlling cholesterol levels could contribute to improved clinical outcomes.

Declaration of competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Ethical approval

The institutional review board/Ethics Committee of Combined Military Hospital (CMH) Rawalpindi approved the current research work under ethical approval number 425.

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None.

CRediT authorship contribution statement

The authors declare their contribution as follows. **Sumera Zaib**: Conceptualization, Methodology, Writing – review & editing original draft, Supervision, Project administration, **Usman Wajid**: Investigation, Methodology, Formal analysis, Writing – review & editing original draft, **Hafiz Saqib Ali:** Investigation, Writing – review & editing original draft, **Muhammad Sohaib Nadeem**: Supervision, Project administration, Resources, **Nehal Rana**: Methodology, Writing – review & editing original draft, **Zainab Zaib:** Formal analysis, Writing – review & editing original draft. All authors have read and agreed to the published version of the manuscript.

Data availability statement

The data generated during and/or analyzed during the current research work are available from the corresponding author on request.

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