World Journal of Advanced Research and Reviews

eISSN: 2581-9615 CODEN (USA): WJARAI

Cross Ref DOI: 10.30574/wjarr

Journal homepage: https://wjarr.com/

(Research Article)

Dyslipidaemia, acute kidney injury, and electrolyte imbalance as a predictor for prostate cancer

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World Journal of Advanced Research and Reviews, 2026, 29(03), 1454-1460

Publication history: Received on 08 February 2026; revised on 20 March 2026; accepted on 23 March 2026

Article DOI: https://doi.org/10.30574/wjarr.2026.29.3.0667

Abstract

Introduction - Prostate cancer is the second most common cause of cancer-related deaths in men and is projected to increase to about half a million deaths by 2030. The burden of prostate cancer is bound to affect resource-poor regions, where treatment options are limited and low-cost community-wide screening is lacking.

Aim - This study assessed and compared the prostate-specific antigen (PSA) and electrolyte levels, kidney function, and lipid profiles of patients with prostate cancer to a control population.

Method - This case–control study involved 60 male participants (30 prostate cancer patients and 30 controls), aged 50–99 years, who were age-matched, with comparable mean ages ($p = 0.4752$) at Nnamdi Azikiwe University. Total PSA level was estimated using an immunoenzymometric method. The electrolyte level was estimated using ion selection, and lipid profiles were estimated spectrophotometrically.

Result - Patients with prostate cancer had significantly higher serum PSA ($p<0.001$), creatinine ($p<0.05$), urea ($p<0.05$), chloride ($p<0.05$), bicarbonate ($p<0.01$), and total cholesterol ($p<0.01$) levels than those in the control group. There was a significant decrease in the HDL levels of participants with prostate cancer compared to the control.

Conclusion - Patients with prostate cancer showed significantly elevated PSA levels, along with evidence of dyslipidaemia, impaired kidney function, and electrolyte imbalance compared with controls. These findings suggest that prostate cancer may be associated with alterations in lipid metabolism, renal function, and electrolyte homeostasis. Therefore, assessment of lipid profile, kidney function parameters, and electrolytes may provide useful supportive clinical information in the evaluation and monitoring of patients with suspected or confirmed prostate cancer, particularly in those with elevated PSA levels.

Keywords: Prostate cancer; Prostate specific antigen; Electrolytes; High-density lipoprotein-cholesterol

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1. Introduction

Prostate cancer (PC) is the second most frequently diagnosed cancer in men and the fifth leading cause of cancer-related deaths among men globally, with over 1.46 million new cases and approximately 396,000 deaths reported in 2022[1]. In Africa, PC is the most prevalent cancer and the leading cause of cancer-related mortality in men[2]. PC is responsible for 13% of all male cancer incidences and 11.3% of all male cancer-associated mortality[3]. Data from GLOBOCAN 2012 indicate that in Africa, the incidence rate of prostate cancer is 23.2 per 100,000, while the mortality rate is 17.0 per 100,000[4]. In Nigeria, PC was ranked as the number one cancer in males, as reported by Ogunbiyi and Shittu (1999), and the other by[5]. It is the most common cancer among Nigerian men, with an age-adjusted incidence and mortality rates of 22.7 and 18.6 per 100,000, respectively[6]. It contributes to 18.2% of all cancer diagnoses and 17.7% of cancer-related deaths in males. Nigeria's large population, estimated at 160 million in 2012, represents a substantial number of men affected by the disease[6]. Despite its high incidence and prevalence, PC has a relatively slow progression from early to advanced disease and can remain undetected for a long time[7]. PC is mostly asymptomatic at its early stage and often requires minimal or no treatment even though complaints such as nocturia and difficulty with urination can be present. Symptoms such as urinary retention and back pain, as the axis skeleton is the most common site of bone metastatic disease, are associated with advanced stages of the disease[8]. Prostate-specific antibodies and prostate biopsies were used to detect and confirm the presence of PC[9], [10]. However, PSA testing can sometimes be misleading, since milder prostate diseases, such as prostatitis and benign prostate hyperplasia, can also elevate PSA levels[11], [12]. This non-sensitivity of PSA as a first step in PC diagnosis can sometimes lead to overdiagnosis, and unnecessary biopsies carry the risk of infection. Therefore, there is a need to explore other potential predictors of PC that can be used alongside PSA testing to confirm the presence of PC.

2. Materials and methods

2.1. Study design

This was a case-control study conducted on prostate cancer patients visiting the Nnamdi Azikiwe University Teaching Hospital (NAUTH). Ethical approval was obtained from the Nnamdi Azikiwe University Teaching Hospital (NAUTH) Ethics Committee.

2.2. Study population

Sixty male participants were recruited for the study. This included 30 subjects with known prostate cancer and 30 healthy subjects (control subjects).

2.3. Inclusion criteria

- Prostate cancer positive subjects
- Healthy subjects (control) above the age of 40 years old
- Those that have no clinical conditions other than prostate cancer.

2.4. Exclusion criteria

- Those below 40years of age
- Subjects presenting with other clinical conditions like diabetes mellitus, renal disease etc.

2.5. Sample collection and processing

A carefully structured questionnaire was used to obtain data, such as age, presence of any other form of complications, and lifestyle. These data were used as an overall eligibility index. Upon completion of the questionnaire, 5 ml of blood was collected from each participant. The blood was dispensed into plain tubes and allowed to clot, centrifuged, separated, aspirated into plain tubes, and kept frozen until assayed for PSA, kidney function, lipid profile, kidney, electrolyte, urea,

2.6. Estimation of total PSA, creatinine, urea, and electrolytes

PSA levels were estimated using an immuno-enzymatic assay. Creatinine levels were determined using the method described by Vasiliades (1976)[13]. Urea content was determined using the technique described by Taylor and Colleague (1992)[14]. The electrolytes were measured using an ion-selective electrode (ISE).

2.7. Lipid profile estimation

Total cholesterol level was determined using a previously described method [15]. The enzymatic hydrolysis method described in [16] was used to determine serum triglyceride levels.

HDL was estimated using the method described in[17], while LDL was determined using the Friedwald equation described in[18].

2.8. Statistical analysis

The data were analyzed using SPSS version 20.0. Student's t-test and Pearson's correlation were used to determine differences and correlations within variables, and values were considered significant at p<0.05. Data are presented as mean ± standard deviation (SD).

3. Result

3.1. PSA, kidney function, electrolyte and lipid profile of study participants.

PSA levels were significantly higher in the PC group than in the control group (P <0.001). Creatinine and urea levels were both higher in participants with PC than in the control group (p=0.006, 0.001). Chloride was significantly higher and bicarbonate was significantly lower in the PC group than in the control group (p=0.003 and p <0.001, respectively). HDL and TC levels were significantly lower and higher, respectively, in participants with PC than in the control (p=0.003, 0.018). The sodium, potassium, and triglyceride levels were not significantly different between the groups.

Table 1 Age distribution of study participants

Group	Frequency (N)	Mean age (Years)	SD	P value
Group A (Prostate cancer)	30	70.47	7.328	0.4752
Group B (Control)	30	69.00	8.436	

This table shows a comparison of the mean ages of the prostate cancer and control groups. years had a mean age of 70.47 ± 7.3 years (SD]), while the control participants had a mean age of 69.00 ± 8.4 years. There was no statistically significant difference in age between the groups (p = 0.4752).

Table 2 The PSA level, creatinine, urea, lipid profile, and electrolytes of both the test and control groups

Variables	Prostate Cancer (mean ± SD)	Control (mean ± SD)	p-value
tPSA (ng/ml)	52.80 ± 46.92	1.630 ± 1.2287	0.000*
Creatinine (μmol/l)	102.19 ± 56.91	69.59 ± 15.937	0.006*
Urea (mmol/l)	6.097 ± 3.3209	3.783 ± 1.1638	0.001*
Na (mmol/l)	143.76 ± 5.076	142.23 ± 1.755	0.126
K (mmol/l)	4.169 ± 0.6735	4.263 ± 0.3528	0.501
Cl (mmol/l)	103.24 ± 4.572	100.43 ± 2.012	0.003*
HCO_3 (mmol/l)	21.83 ± 2.740	4.17 ± 1.984	0.000*
TC (mmol/l)	4.9931 ± 0.95166	4.7237 ± 0.70704	0.018*
TG (mmol/l)	1.2238 ± 0.39803	1.0810 ± 0.25698	0.106
HDL-c (mmol/l)	1.0686 ± 0.26012	1.2990 ± 0.31460	0.003*
LDL-c (mmol/l)	2.5664 ± 0.79660	2.9317 ± 0.69910	0.066

Data were analyzed using Student's t-test. Values are presented as mean ± standard deviation and were considered significant at p<0.05.

tPSA, Total Prostate Specific Antigen, Na, sodium; K, potassium; Cl = Chloride, HCO_3 = Bicarbonate, TC = Total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol.

Table 3 Correlation of PSA levels to electrolytes, kidney, and lipid parameters in participants with PC

Correlating Pair	Participants with PC (r, p-value)
tPSA (ng/ml) – Creatinine (µmol/l)	0.597, 0.001*
tPSA (ng/ml) – Urea (mmol/l)	0.603, 0.001*
tPSA (ng/ml) – Na (mmol/l)	0.087, 0.655
tPSA (ng/ml) – K (mmol/l)	-0.101, 0.603
tPSA (ng/ml) – Cl (mmol/l)	0.233, 0.224
tPSA (ng/ml) – HCO_3 (mmol/l)	-0.363, 0.053
tPSA (ng/ml) – TC (mmol/l)	-0.169, 0.380
tPSA (ng/ml) – TG (mmol/l)	-0.014, 0.943
tPSA (ng/ml) – HDL-c (mmol/l)	-0.150, 0.438
tPSA (ng/ml) – LDL-c (mmol/l)	-0.150, 0.437

tPSA, total prostate-specific antigen; Na, sodium; K, potassium; Cl = Chloride, HCO_3 = Bicarbonate, TC = Total cholesterol; TG, triglyceride; HDL-c, high-density lipoprotein cholesterol; LDL-c, low-density lipoprotein cholesterol.

The data were analyzed using Pearson's correlation. Values are presented as mean ± standard deviation and were considered significant at $p<0.05$. This table shows the correlation of total PSA levels with creatinine, urea, sodium, potassium, chloride, bicarbonate, total cholesterol, triglyceride, HDL, and LDL levels. PSA levels showed a significant positive correlation with creatinine ($r=0.597$, $p = 0.001$) and urea ($r = 0.603$, $p = 0.001$) levels. No significant correlation was observed between the total PSA level and any other assessed parameter.

4. Discussion

The global burden of prostate cancer (PC) is estimated to increase by 2030 to 1.7 million new cases and approximately half a million deaths[19]. This burden is bound to be heavier in resource-poor regions, where the incentives for early screening are small, and available treatment options are limited[20]. There is a need to explore more options that could predict the development or progression of PC, if its burden must be managed. This study assessed and compared the lipid profile, electrolyte levels, kidney function, and PSA levels of patients with PC to those of a control population.

The results showed that PSA levels were significantly higher in the PC group than in the control group ($p<0.001$) (Table 2). This finding is expected and unsurprising, given that PSA is a well-known biomarker for PC diagnosis. Generally, a PSA level of 4.0ng/mL or lower is considered normal, and this corroborate the mean PSA levels of 52.8ng/mL and 1.63ng/mL observed in participants with PC and the control group respectively. Participants with PC had significantly higher creatinine ($p=0.006$) and urea ($p=0.001$) levels than those in the control group. It is possible that increased creatine and urea, an indication of kidney dysfunction, is a direct complication of PC, since some anti-cancer treatments such as androgen deprivation therapy have been shown to increase the risk of acute kidney injury[21]. However, studies have shown a bidirectional relationship between cancer and CKD[22], [23], [24]. This indicates that cancer or chemotherapy can induce kidney damage, just as renal disease can be a risk factor for cancer. This relationship was demonstrated in a study that assessed PSA levels in patients with end-stage renal disease (ESRD). The study found significantly increased levels of PSA in patients with ESRD[25], suggesting that kidney dysfunction might be directly involved in PC pathogenesis. Correlation analysis also showed a significant positive correlation between serum PSA, creatinine, and urea levels (table 2). Total cholesterol (TC) and HDL were significantly increased and decreased, respectively, in the PC group compared to the control group ($p=0.018$, 0.003). Increased TC and decreased HDL levels indicate the presence of dyslipidemia, which is a significant risk factor for cardiovascular diseases, such as coronary heart disease. This finding is in line with previous studies, which revealed that approximately two-thirds of patients with PC are at high cardiovascular risk[26], [27]. Although the causative relationship between PC and CVDs remains unclear, studies have shown that patients treated for cancer are 4-6 times more likely to die from cardiovascular illness[28], [29]. Some authors have associated the increased CVDs risk in men with PC to Androgen deprivation therapy (ADT), as decreased testosterone due to ADT has been shown to disrupt the normal lipid profile[30], [31]. Interestingly, however, the PSA levels in our study did not show any significant correlation with the lipid profile of participants with PC (table 3). The mean serum chloride and bicarbonate levels in participants with PC were significantly higher than those in the control group (p=.003, p<0.001, respectively). Electrolyte imbalance is a common side effect of certain antineoplastic drugs[32], [33], and this could explain the hyperchloremia observed in patients with PC.

Overall, participants with prostate cancer, in addition to having significantly elevated PSA levels, also showed signs of dyslipidemia, acute kidney injury, and electrolyte imbalance. These disorders appear to be associated with comorbidities that can be used alongside PSA to predict the progression and onset of PC. PSA screening alone sometimes fails to discriminate prostate cancer from other mild prostate disorders, often leading to overdiagnosis[34]. There is a need to supplement PSA screening with other noninvasive, low-cost tests that can differentiate PC from other benign prostate diseases. Our study provides preliminary evidence that lipid profile assessment, electrolyte tests, and kidney function tests can be beneficial tests to confirm the presence of PC in patients with elevated PSA levels.

4.1. Strengths and Limitations

A major strength of this study is its comprehensive assessment of multiple biochemical parameters, namely lipid profile, kidney function markers, electrolyte levels, and PSA, in a single cohort of men with and without prostate cancer. This approach provides a broader understanding of the metabolic disturbances that may accompany or predict prostate cancer than PSA alone. By comparing prostate cancer patients with age-matched controls (Table 1), this study enhances the validity of the observed differences and helps establish clinically meaningful associations. Another strength is the integration of correlation analysis, which allowed the study to explore possible mechanistic associations between PSA levels, kidney dysfunction, and metabolic imbalance. These findings also contribute to the limited evidence available from sub-Saharan Africa regarding biochemical comorbidities of prostate cancer, particularly in areas with rising cancer incidence but limited diagnostic resources. This enhances the relevance of this study for early detection and risk-stratification strategies in low- and middle-income countries.

Despite its strengths, this study has several limitations. First, its cross-sectional design restricts causal interpretation. Although associations were identified between dyslipidemia, kidney function biomarkers, electrolyte imbalance, and PSA levels, the temporal or causal direction of these relationships cannot be established. Longitudinal designs are needed to determine whether these biochemical abnormalities precede prostate cancer development or arise as complications of the disease or treatment.

Second, the study relied on hospital-based sampling, which may have introduced selection bias, as individuals presenting at tertiary facilities may differ from the general population in disease severity, comorbidities, or access to care. This may limit the generalizability of our findings to community-based populations.

5. Conclusion

Dyslipidemia, hyperchloremia, and chronic kidney disease are present in patients with prostate cancer and could be potential predictors of the disease, particularly in those with above normal PSA levels. To date, no causal relationship has been established between prostate cancer and any of the identified diseases. Further studies are needed to bridge these gaps to fully understand the mechanism and direction of this relationship.

Compliance with ethical standards

Disclosure of conflict of interest

The authors declare no competing interests.

Statement of ethical approval

This entire study procedure was reviewed and approved by the Nnamdi Azikiwe University Teaching Hospital (NAUTH) Health Research Ethics Committee (Approval Number: NAUTH/CS/66/VOL.7/92). All procedures involving human participants adhered to the ethical standards of the institutional review board and principles outlined in the Declaration of Helsinki.

Statement of informed consent

Written informed consent was obtained from all participants prior to their enrollment in the study. Participation was voluntary, and the confidentiality of all personal and clinical information was strictly maintained throughout the study.

Authors' contributions

KCE and UA conceived and designed the study; KCE performed the experiments and collected the data; NPO and KCE analysed the data and wrote the paper. All authors have read and approved the final manuscript.

Availability of data and materials

Data presented in this study are available upon request from the first author (kingsley.eboh49@gmail.com).

References

[1] Schafer EJ, Laversanne M, Sung H, Soerjomataram I, Briganti A, Dahut WL, et al. Recent patterns and trends in global prostate cancer incidence and mortality: an update. Eur Urol. 2025;87(3):302–313.

[2] Marais B, Klopper G, John J. Prostate cancer perspective: Africa versus the world. S Afr Med J. 2024;114(4):21–25.

[3] Akinremi TO, Ogo CN, Olutunde AO. Review of prostate cancer research in Nigeria. In: Infectious Agents Cancer. Springer; 2011. p. 1–4.

[4] Ferlay J, Soerjomataram I, Dikshit R, Eser S, Mathers C, Rebelo M, et al. Cancer incidence and mortality worldwide: sources, methods and major patterns in GLOBOCAN 2012. Int J Cancer. 2015;136(5):E359–E386.

[5] Mohammed AZ, Edino ST, Ochicha O, Gwarzo AK, Samaila AA. Cancer in Nigeria: a 10-year analysis of the Kano cancer registry. Niger J Med. 2008;17(3):280–284.

[6] Agalliu I, Adebiyi AO, Lounsbury DW, Popoola O, Jinadu K, Amodu O, et al. The feasibility of epidemiological research on prostate cancer in African men in Ibadan, Nigeria. BMC Public Health. 2015;15:425.

[7] Giovannucci E, Liu Y, Platz EA, Stampfer MJ, Willett WC. Risk factors for prostate cancer incidence and progression in the Health Professionals Follow-Up Study. Int J Cancer. 2007;121(7):1571–1578.

[8] Rawla P. Epidemiology of prostate cancer. World J Oncol. 2019;10(2):63–89.

[9] Zappa M, Puliti D, Hugosson J, Schröder FH, van Leeuwen PJ, Kranse R, et al. A different method of evaluation of the ERSPC trial confirms that prostate-specific antigen testing has a significant impact on prostate cancer mortality. Eur Urol. 2014;66(2):256–262.

[10] Descotes JL. Diagnosis of prostate cancer. Asian J Urol. 2019;6(2):129–136.

[11] David MK, Leslie SW. Prostate-specific antigen. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2024.

[12] Orugbo VP, Ntaji M. Distribution of age-specific prostate-specific antigen profiles in men between 40 and 80 years tested in a urology clinic in Oghara, Delta State, Nigeria. Afr J Health Saf Environ. 2022;3(1):11–18.

[13] Vasiliades J. Reaction of alkaline sodium picrate with creatinine: I. Kinetics and mechanism of formation of the mono-creatinine picric acid complex. Clin Chem. 1976;22(10):1664–1671.

[14] Taylor AJ, Vadgama P. Analytical reviews in clinical biochemistry: the estimation of urea. Ann Clin Biochem. 1992;29(Pt 3):245–264.

[15] Röschlau P, Bernt E, Gruber W. Enzymatic determination of total cholesterol in serum. Z Klin Chem Klin Biochem. 1974;12(5):226–230.

[16] Tietz NW. Clinical guide to laboratory tests. 2nd ed. Philadelphia: W.B. Saunders; 1990. p. 566.

[17] Burstein M, Scholnick HR, Morfin R. Rapid method for the isolation of lipoproteins from human serum by precipitation with polyanions. J Lipid Res. 1970;11(6):583–595.

[18] Bairaktari E, Hatzidimou K, Tzallas C, Vini M, Katsaraki A, Tselepis A, et al. Estimation of LDL cholesterol based on the Friedewald formula and on apolipoprotein B levels. Clin Biochem. 2000;33(7):549–555.

[19] Alvarez C, Virani S, Meza R, Rozek LS, Sriplung H, Mondul AM. Current and future burden of prostate cancer in Songkhla, Thailand: analysis of incidence and mortality trends from 1990 to 2030. J Glob Oncol. 2018;4:1–11.

[20] Ugwumba FO, Nnabugwu II. Prostate cancer characteristics: a descriptive analysis of clinical features at presentation in the last decade in a Black African community. Ann Afr Med. 2022;21(2):153–159.

[21] Lapi F, Azoulay L, Niazi T, Yin H, Benayoun S, Suissa S. Androgen deprivation therapy and risk of acute kidney injury in patients with prostate cancer. JAMA. 2013;310(3):289–296.

[22] Małyszko J, Bamias A, Danesh FR, Dębska-Ślizień A, Gallieni M, Gertz MA, et al. KDIGO controversies conference on onco-nephrology: kidney disease in hematological malignancies and the burden of cancer after kidney transplantation. Kidney Int. 2020;98(6):1407–1418.

[23] Hu M, Wang Q, Liu B, Ma Q, Zhang T, Huang T, et al. Chronic kidney disease and cancer: inter-relationships and mechanisms. Front Cell Dev Biol. 2022;10:748964.

[24] Stengel B. Chronic kidney disease and cancer: a troubling connection. J Nephrol. 2010;23(3):253–262.

[25] Khochikar MV. Is incidence of prostate cancer higher in patients with end-stage renal disease? Indian J Urol. 2007;23(1):35–37.

[26] Leong DP, Fradet V, Shayegan B, Duceppe E, Siemens R, Niazi T, et al. Cardiovascular risk in men with prostate cancer: insights from the RADICAL PC study. J Urol. 2020;203(6):1109–1116.

[27] Zhanghuang C, Wang H, Wang J, Li L, Li J, Hao Z, et al. Chemotherapy and heart-specific mortality in elderly men with prostate cancer: a propensity score matching analysis. PLoS One. 2025;20(4):e0312345.

[28] Liu D, Ma Z, Yang J, Zhao M, Ao H, Zheng X, et al. Prevalence and prognostic significance of cardiovascular disease in cancer patients: a population-based study. Aging (Albany NY). 2019;11(18):7948–7960.

[29] Cleary S, Rosen S, Gilbert D, Langley R. Cardiovascular health: an important component of cancer survivorship. BMJ Oncol. 2023;2:e000090.

[30] Hu JR, Duncan MS, Morgans AK, Brown JD, Meijers WC, Freiberg MS, et al. Cardiovascular effects of androgen deprivation therapy in prostate cancer: contemporary meta-analyses. Arterioscler Thromb Vasc Biol. 2020;40(3):e55–e64.

[31] Kim J, Freeman K, Ayala A, Mullen M, Sun Z, Rhee JW. Cardiovascular impact of androgen deprivation therapy: from basic biology to clinical practice. Curr Oncol Rep. 2023;25(9):965–977.

[32] Verzicco I, Regolisti G, Quaini F, Bocchi P, Brusasco I, Ferrari M, et al. Electrolyte disorders induced by antineoplastic drugs. Front Oncol. 2020;10:779.

[33] Turcotte A, Achi S, Mamlouk O, Mandayam S. Electrolyte disturbances in cancer patients. Curr Opin Nephrol Hypertens. 2022;31(5):425–434.

[34] Kombathula S, Deepak V, Chappantakath R, Sejpal J, Jasurbek B, Azamat B, et al. Prostate-specific antigen testing in prostate cancer detection: a systematic review of sensitivity, specificity, and false-positive influences. Int J Contemp Pathol. 2025;11:4–13.