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ORIGINAL RESEARCH ARTICLE

Discrepancy between PSA Levels and Gleason Score in Prostate Cancer from Northern Nigeria: A Histopathological Analysis

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ABSTRACT

An extensively utilized tumor marker for prostate cancer is prostate-specific antigen (PSA). It's well known that PSA is a marker specific to the prostate rather than a disease. The study aims to determine the relationship between prostate-specific antigen and various prostatic lesions in some states in North-western Nigeria. This study included 224 samples from the histopathology laboratory of two teaching hospitals in North-western Nigeria. Representative tissue was obtained from the samples, and paraffin-embedded tissue blocks were made, sectioned, and stained with hematoxylin and eosin. The slides underwent light microscopy examination for a final assessment and diagnosis. An Electrochemiluminescence immunoassay was used to measure serum PSA levels. The study analyzed 224 samples, revealing benign prostatic hyperplasia in 54.9% of cases, while prostate adenocarcinoma was the most common malignancy, accounting for 35.7% of cases. Normal PSA levels were found in 17.0% of BPH cases and 11.2% of prostate cancer cases. Benign lesions (54.9% BPH) showed elevated PSA (>20 ng/ml) in 27.7% of cases, while 58.8% of malignancies had PSA >20 ng/ml. The findings show a statistically significant correlation ($p < 0.05$) between prostatic lesions and serum total PSA levels, and no significant association between PSA levels and Gleason scores ($r = 0.098$, $p > 0.05$). Serum PSA levels can rise due to both benign and malignant tumors, and the likelihood that a benign disease would progress to a malignant one increases with increased PSA levels. Adults over 40 must consider preventative steps like prostate cancer screening.

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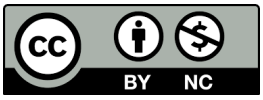

INTRODUCTION

Especially in affluent nations, prostate cancer (PCa) is the most prevalent urological malignancy and causes a significant health burden worldwide (Huang *et al.,* 2023). About 14.1% and 6.8% of all cancer cases and fatalities in men in 2020 were due to prostate cancer (Huang *et al.,* 2023). An indication of a malignant tumor of the prostate is prostate cancer. Most of these malignant neoplasms originate from the prostatic epithelium and are carcinomas (Humphrey, 2017). Prostate cancer (PCa) is the second most common cancer in men worldwide, causing significant illness and death. It's particularly impactful for men of African descent, who often experience higher morbidity rates (Iheanacho & Enechukwu, 2025).

The exact cause of the disease remains relatively unknown (Sung *et al.,* 2021). About 9% of people with prostate cancer have a family history of cancer, indicating a substantial correlation between prostate cancer risk factors and a family history of any disease. Certain genetic mutations (e.g., BRCA1 and BRCA2), conditions (e.g., Lynch syndrome), and the number of affected persons, degree of link, and age at illness start are taken into account when calculating family risk (Rebello *et al.,* 2021).

Human kallikrein 3 (hK3), often known as PSA, is a glandular kallikrein highly expressed in the prostate and encoded by the KLK3 gene. It is used as a marker to track the success of prostate cancer treatment and to monitor response to it (Filella & Foj, 2016). Prostate-specific antigen (PSA) is released into the bloodstream as a result of pathological processes that compromise cell integrity; in other words, internal prostate processes like inflammation, tumors, and hyperplasia can raise serum PSA levels (Stimac *et al.,* 2014; Gurumurthy *et al.,* 2015).

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The adult prostate gland is divided into four distinct zones: peripheral, central, transitional, and periurethral. Different types of lesions tend to occur in each zone, with most benign growths (hyperplasia) occurring in the transitional zone and most cancers (carcinomas) originating in the peripheral zone (Sung *et al.,* 2021).

Elevated PSA plasma levels (PSA > 4 ng/mL) are a common marker for prostate cancer. However, a tissue biopsy is the gold standard for confirming the presence of cancer because increased PSA has also been seen in men without cancer (Rawla, 2019). The Gleason grading system, the most used histological grading technique for prostatic adenocarcinoma, is used to assess the aggressiveness of prostate cancer (PCa). It is based on the architectural characteristics of prostate cancer cells. The Gleason score provides a significant prognostic indicator and exhibits a strong correlation with clinical behavior (Graham *et al.,* 2008). Furthermore, coupled with stage, age, and PSA, this score is a crucial factor in therapy decision-making (Graham *et al.,* 2008). The epithelial lining of prostatic acini and ducts is the cells responsible for producing PSA. For prostate cancer, PSA is the recommended serum marker due to its excellent specificity for prostate tissue. However, PSA is unique to prostate tissue but not to prostate cancer; it is frequently detected at abnormally high levels in benign and typical changes of the prostate, including BPH and other non-neoplastic prostatic lesions. The overlap in PSA results between individuals with organ-confined prostate cancer and those with BPH raises doubts about the use of PSA alone in early detection of prostate cancer (Maru *et al.,* 2014).

A wide variety of markers, such as prostate-specific membrane antigen (PSMA), prostate stem cell antigen (PSCA), ETS-related gene product (ERG), prostein (p501s), androgen receptor (AR), citric acid, polymorphonuclear (PMN) cell elastase, and glutathione S-transferase-1 (GSTP-1), are available for the detection of prostate cancer. Because PSA is a protein produced by the prostate gland and is present in high amounts, it is preferred to measure. Finding tumors that have spread outside of the prostate gland is more likely when the PSA level in the blood. While PSA-Gleason correlations are documented globally, regional variations in Nigeria remain poorly characterized, particularly in the North-western states, where diagnostic access is limited. This study addresses this gap by analyzing histopathologically confirmed cases from Kano and Zaria. Thus, our goal is to assess the correlation between prostate-specific antigen levels, Gleason score, and different prostatic lesions in Northern Nigerian individuals who may have prostate cancer.

METHODS

Study design and location

This prospective cross-sectional study was conducted at the Departments of Histopathology and Chemical Pathology of Aminu Kano Teaching Hospital, Kano, and Ahmadu Bello University Teaching Hospital (ABUTH), Zaria, both located in north-western Nigeria, from September 2019 to February 2021.

Sample Size Determination

The sample size was calculated using the formula provided by Niang *et al.* (2006).

$$n = \frac{Z^2pq}{d^2}$$

Where:

n= number of samples

Z= confidence interval of 95% = 1.96

p= Prevalence of Prostate Cancer in Kano = 16.5% (Mohammed *et al.,* 2003)

q= (1-p)

d= Error margin taken to be 5% (0.05)

$$n = \frac{Z^2pq}{d^2}$$

$[(1.96)^2 \times 0.165 \times (1-0.165)]/ 0.05 \times 0.05 = 211.7$

The minimum sample size was therefore approximately 212.

Blood sample collection and analysis

A simple vacutainer blood container was filled with about 5 ml of blood drawn from each subject using a syringe and needle. The blood was centrifuged for ten minutes at 5000 rpm after being given time to clot. After that, sterile cryovials were used to extract the clear, non-haemolyzed serum (Delta lab, Barcelona, Spain). The serum was kept in storage at -20 degrees Celsius while it was being examined.

Biological Principles of the Procedure

The ARCHITECT Total PSA assay is a two-step immunoassay created to detect total PSA levels (including both free PSA and PSA complexed with alpha-1-antichymotrypsin) in human serum. It employs Chemiluminescent Microparticle Immunoassay (CMIA) technology with adaptable assay protocols known as Chemiflex (Abbott, Abbott Park, Illinois, USA). In the initial step, the sample is mixed with anti-PSA-coated paramagnetic microparticles, allowing PSA in the sample to bind to these particles. After washing, an anti-PSA acridinium-labeled conjugate is introduced in the second step. Following this, Pre-Trigger and Trigger Solutions are added to the reaction mix, and the resulting chemiluminescent reaction is measured in relative light units (RLUs). There is a direct correlation between the total PSA quantity in the sample and the RLUs detected

by the ARCHITECT i* optical system. The precision of the ARCHITECT Total PSA assay is ≤8%, demonstrating good accuracy (Gill et al., 2018).

Biopsy collection, processing, and examination

Prostatic biopsies were collected from 224 patients for histopathological analysis. The tissues were fixed in 10% formalin immediately after collection and then processed using an automated tissue processing machine (Leica, Lakewood Ranch, Florida, USA). Sections approximately 5 µm thick were cut from paraffin-embedded tissue blocks with a Leica RM-2125 RTS rotary microtome (Leica, Lakewood Ranch, Florida, USA). The sections were then stained with Haematoxylin and Eosin (H&E). Light microscopy was used to analyze the sections. The diagnosis of adenocarcinoma, prostatitis, prostatic intraepithelial neoplasia (PIN), and benign prostatic hyperplasia (BPH) was made according to the previous study. (Montironi et al., 2016). Adenocarcinoma was graded using the WHO-adopted Gleason scoring methodology (Montironi et al., 2016).

Statistical analysis

Data analysis was performed using IBM SPSS Statistics software (version 25.0), involving descriptive statistics (mean ± standard deviation) and Pearson's Chi-square test to examine relationships between variables. Pearson's correlation coefficient was employed to test the relationship between PSA and Gleason Score, with statistical significance set at $p < 0.05$.

RESULTS

The frequency of prostatic lesions among different age groups

The study included 224 subjects, the majority of whom (36.7%) were between the ages of 61 and 70. On the other hand, the least percentage (0.9%) of participants were older than 90. Table 1 displays the distribution of prostatic lesions by age. 144 (64.2%) of the 224 biopsy specimens showed benign lesions upon histopathological analysis. The most prevalent benign lesion, involving 123 (54.9%) of the individuals, was BPH. BPH with PIN (2.2%, n = 5) and BPH with CP (7.1%, n = 16) were additional benign lesions.

Frequency Distribution of various prostatic lesions and histologic characteristics

Prostatic adenocarcinomas were the most commonly detected malignancy, representing 35.7% (80 cases) of the study population. The microscopic and histologic characteristics of the lesions are illustrated in Figures 1 and 2.

Association between total PSA levels and various prostatic lesions

A total of 21 (17.0%) BPH patients had normal serum total PSA levels (<4 ng/ml), whereas BPH with prostatitis showed none. Only 1 (20.0%) subject with BPH + PIN and 9 (11.2%) cases of prostatic adenocarcinoma had normal PSA levels. Serum total PSA was found to be somewhat elevated in 10 (12.5%) prostatic adenocarcinoma individuals, 37 (31.1%) BPH patients, 8 (50.0%) BPH + CP patients, and 1 (20.0%) BPH + PIN participants. 34 patients (27.7%) with BPH and 47 patients (58.8%) with PCa had elevated blood total PSA levels (>20 ng/ml).

As shown in Table 2, there was a statistically significant correlation ($\chi2 = 31.34$, $p = 0.00$) between the PSA levels and prostatic lesions of the study participants.

Association between serum total PSA with Gleason Score in prostatic adenocarcinoma

As shown in Table 3, the only cancer discovered in the research participants was adenocarcinoma. Among patients with serum total PSA values >20 ng/ml, 48 (60%) had Gleason scores of ≤6, 7, or ≥8. No statistically significant association ($r = 0.098$, $\chi2 = 6.97$, $P = 0.32$) was found between serum total PSA levels and Gleason score. Gleason's grading of prostate cancer revealed that patients with a score of 6 had a frequency of 19 (23.7%), while those with a score of 7 were the most common (27, 33.8%). Patients with a score of 10 were the least frequent (3, 3.8%), as shown in Table 4.

DISCUSSION

Prostate cancer accounted for 1,276,106 new cases and 358,989 fatalities (3.8% of all male cancer-related deaths) in 2018, making it the second most common disease in males globally (after lung cancer) (Bray *et al.,* 2018; Ferlay *et al.,* 2021).

Table 1: The frequency of prostatic lesions among different age groups

Age Group	BPH n (%)	BPH+CP n (%)	BPH+PIN n (%)	PCA n (%)	Total N (%)
41 – 50	7 (5.7)	0 (0.00)	0 (0.00)	2 (2.50)	9 (4.0)
51 – 60	33(26)	5 (31.25)	0 (0.00)	27 (33.75)	65 (29.0)
61 – 70	47(38.2)	6 (37.50)	2 (40.00)	27 (33.75)	82 (36.7)
71 – 80	30 (24.3)	4 (25.00)	1 (20.00)	22 (27.50)	57 (25.5)
81 – 90	5 (4.1)	1 (6.25)	1 (20.00)	2 (2.50)	9 (4.0)
>90	1 (0.9)	0 (0.00)	1 (20.00)	0 (0.00)	2 (0.9)
TOTAL	123 (100)	16 (100)	5 (100)	80 (100)	224 (100)

n: number of samples.

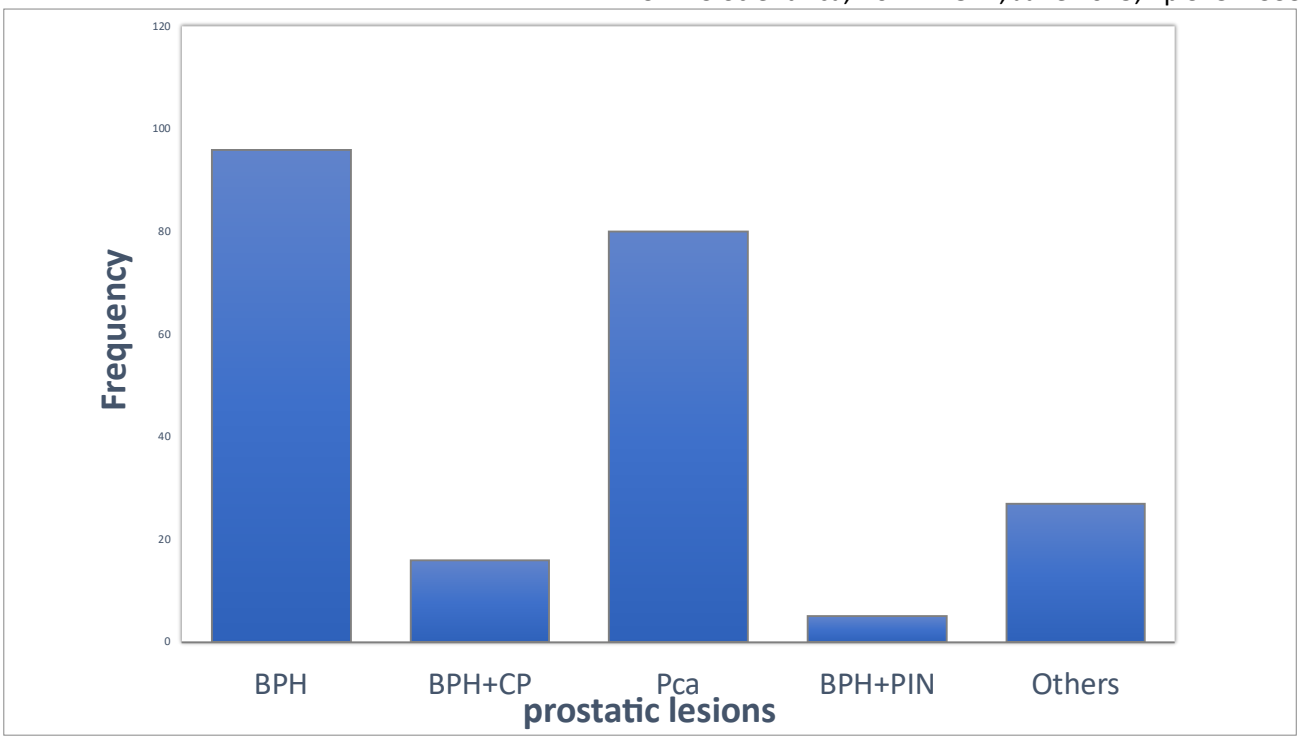

Figure 1: Frequency distribution of various prostatic lesions

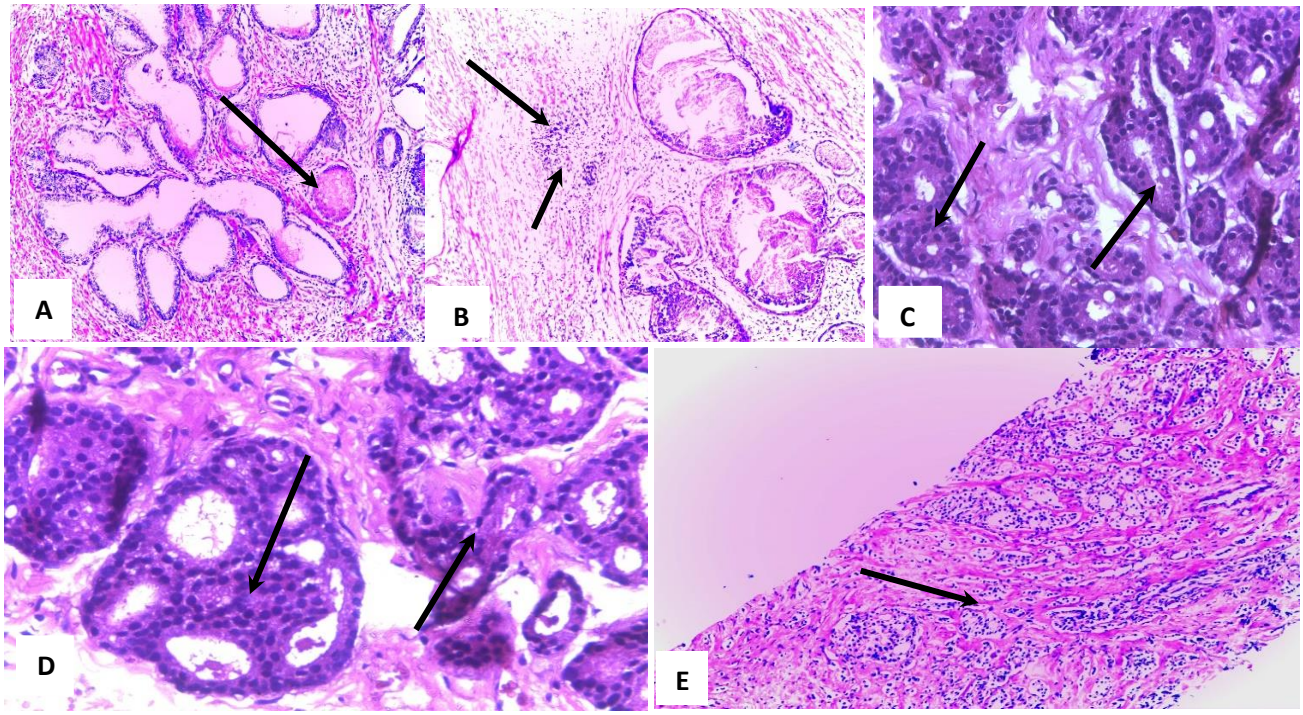

Figure 2: Photomicrograph of Haematoxylin and eosin-stained tissue showing (A) Benign Prostatic Hyperplasia showing acini lined by hyperplastic columnar epithelium and containing eosinophilic corpus amylaceum dispersed in a fibromuscular stroma. H and E x100 (B) Benign Prostatic Hyperplasia with chronic prostatitis demonstrating infiltrates of lymphocytes and plasma cells (arrows) in a fibromuscular stroma containing hyperplastic acini. H and E x100. (C) Prostatic adenocarcinoma showing closely packed, small-sized malignant acini lying back to back with scanty intervening stroma. Gleason score of 7 (3+4). H and E x400. (D) Prostatic adenocarcinoma demonstrating closely packed malignant acini exhibiting cribriform bridging of epithelium. Gleason score of 7 (4+3). H and E x400. (E) Core needle biopsy showing sheets of malignant epithelial cells infiltrating a fibromuscular stroma with a focal cribriform pattern characteristic of poorly differentiated adenocarcinoma. Gleason score of 9 (5+4).

Globally, prostate cancer incidence and fatality rates increase with age; the average age at diagnosis is 66. Of the prostatic lesions identified in this investigation, BPH was the most common, occurring in 123 cases with a frequency of 54.9%. Similarly, our findings align with those of a study conducted in Calabar, Southern Nigeria,

which found that 76.3% of subjects had prostatic lesions (Umezurike *et al.,* 2006). In contrast, previous studies reported prevalence rates of BPH as 77.6% and 75.9% (Mohammed *et al.,* 2003; Abubakar *et al.,* 2019). Conversely, Banerjee and colleagues (Banerjee *et al.,* 2016) reported prevalence rates of 38.8%, 28.75%, and 7.5% for BPH, BPH with prostatitis, and prostatitis without BPH, respectively. The possible explanation for these disparities is a lack of knowledge on methods to enhance prostate health, coupled with the state of health systems in developing countries, inadequate prostate cancer diagnostics, socioeconomic status, cultural confinements, and tumor biological behaviour.

Prostatic adenocarcinoma was shown to be highly prevalent in the current investigation, with 35.7% (80 cases), which was similar to the findings of a previous study, 37.4% (Obiorah & Nwosu, 2011). Conversely, lower prevalence rates were found in the same study location, 22.4% and 23.3% (Mohammed *et al.,* 2003; Abubakar *et al.,* 2019), respectively. In contrast to our results, a study by Hirachand *et al.* (2017) found that the incidence of prostatic adenocarcinoma was 10.16%. We found that the prevalence of BPH + PIN lesions was lower in the current study. This is under the findings of Banerjee *et al.* (2016), where participants with PIN represented roughly 10%. The higher life expectancy in wealthy nations may be somewhat to blame for this discrepancy. Prostate cancer incidence has increased globally, particularly in low- and middle-income nations. As a result, governments and policymakers must work more closely together to allocate funds for bolstering health systems and creating policies to address the threat of prostate cancer. Moreover, recently, there have been ongoing efforts in raising the global awareness of the disease, which has yielded positive health outcomes among older male adults in developed countries.

Our study found that prostatic lesions were most prevalent (36.7%) in individuals aged 61-70 years, consistent with previous studies by Nwafor *et al.* (2015) and Banerjee *et al.* (2016). This age-related trend may be attributed to increased exposure to environmental carcinogens and shared lifestyle habits. However, Pudasaini *et al.* (2019) reported a slightly older peak age range (70-79 years) for prostate cancer. Generally, prostate cancer risk increases after age 50, with some guidelines recommending screening at age 40 for high-risk individuals and considered as an age-related disease, with increasing age associated with an increased risk, as previously reported by Farmer (2008) and the American Cancer Society (2015). Men under 40 are far less likely to be diagnosed than men over 65, who account for more than two-thirds of all cases. Prostate cancer is notably 50% common in males between the ages of 70 and 80, according to biopsy research (Crawford, 2003). Prostate tissue dynamics, such as an imbalance between cell proliferation and death, may be the cause of this age-related tendency, which might be linked to an increasing prevalence of prostatic lesions with aging.

PSA values < 4 ng/ml were seen in the majority of individuals with benign prostatic lesions (BPH, BPH + PIN), as well as in some patients with prostatic adenocarcinoma. In contrast, 58.3% of BPH participants and none of the cancer subjects had normal PSA levels, according to Pudasaini *et al.* (2019). According to Banerjee et al. (2016), benign cases (BPH, BPH + prostatitis, and prostatitis) with PSA values between 0 and 7 ng/ml were reported. Our study revealed that 58.8% of adenocarcinoma cases had PSA levels > 21 ng/ml, which is different from Banerjee et al. (2016), who discovered that 20% of adenocarcinoma patients had PSA > 20 ng/ml and 40% had PSA > 21 ng/ml.

Table 2: Association between total PSA levels and various prostatic lesions

PSA level (ng/ml)	BPH n (%)	BPH+CP n (%)	BPH+PIN n (%)	PCA n (%)	Total N (%)
0 – 4	21 (17.0)	0 (0.00)	1 (20.0)	9 (11.2)	31 (13.9)
4.1 – 10	37 (31.1)	8 (50.0)	1 (20.0)	10 (12.5)	56 (25.0)
10.1 – 20	31 (25.2)	5 (31.2)	0 (0.0)	14 (17.5)	50 (22.3)
>20	34 (27.7)	3 (18.8)	3 (60.0)	47 (58.8)	87 (38.8)
Total	123 (100)	16 (100)	5 (100)	80 (100)	224(100)

χ^2=31.34, p<0.05

Table 3: Association between serum total PSA with Gleason Score in prostatic adenocarcinoma

PSA level (ng/ml)	≤ 6	Gleason Score: 7	≥ 8	Total(%)
0 – 4	4 (21.05)	0 (0.00)	4 (11.7)	8(10.0)
4.1-10	3 (15.70)	4 (14.8)	3 (8.9)	10(12.5)
10.1–20	3 (15.70)	6 (22.2)	5 (14.7)	14(17.5)
>20	9 (47.3)	17(63.0)	22 (64.7)	48(60.0)
Total	19 (100)	27 (100)	34 (100)	80 (100)

X^2=6.97, r = 0.098, P>0.05, which is statistically insignificant; therefore, PSA and Gleason score are unrelated.

In the current investigation, 34 (27.7%) of the BPH patients had a severe rise of serum PSA levels (>20 ng/ml), while Pudasaini and colleagues (2019) observed 0 (0.0%) instances with a total PSA level over 20 ng/ml. The patient's age may have an impact on serum PSA levels, and several disease processes, including prostatitis, PIN, and prostate cancer, can cause the protective layers between the prostatic lumen and capillaries to be compromised, raising serum PSA levels. According to research by Banerjee *et al.* (2016), when glandular

epithelium disruption is present, BPH and prostatitis are linked to elevated PSA. PSA levels can rise in non-cancerous situations such as benign prostatic hyperplasia, inflammation, and diagnostic and surgical operations, which can cause cancerous tissues to seep into the blood. This is another reason why PSA levels in serum might rise. Particularly in cases of prostatic carcinoma, when PSA is utilized as a screening test, these disorders might resemble cancer and make identification more difficult (Lakhey *et al.,* 2010).

Table 4: The distribution of the Gleason Score among different age groups

	Age groups						
GS	41 – 50	51 – 60	61 - 70	71 – 80	81 – 90	Total	N (%)
6	1 (50.00)	5 (17.9)	4 (15.3)	9 (40.9)	0 (0.00)	19 (23.7)	
7	0 (0.00)	11 (39.2)	9 (34.6)	6 (27.2)	1 (50.00)	27 (33.8)	
8	1 (50.00)	5 (17.9)	9 (34.6)	3 (13.6)	1 (50.00)	19 (23.7)	
9	0 (0.00)	6 (21.4)	3 (11.53)	3 (13.6)	0 (0.00)	12 (15.0)	
10	0 (0.00)	1 (3.6)	1 (3.84)	1 (4.5)	0 (0.00)	3 (3.8)	
Total	2 (100)	28 (100)	26 (100)	22 (100)	2 (100)	80 (100)	

χ^2=7.312*; P*=0.503; where GS is the Gleason score,

As reported by Anunobi *et al.* (2011), males in Lagos, southern Nigeria, frequently have high serum PSA levels (>10 ng/ml) due to prostatitis with BPH, which is consistent with the results of our investigation. Prostate cancer, however, has been demonstrated to occur in Caucasian men with serum PSA levels more than 10 ng/ml (Schröder *et al.,* 2001). Although elevated blood PSA levels in benign prostatic illnesses have been documented in the United States, they seldom surpass 30 ng/ml (Dalton, 1989; Neal *et al.,* 1992). Because most benign prostatic lesions in our environment have elevated serum PSA, these factors can lead to diagnostic challenges with prostate cancer detection programs that rely only on serum PSA as a screening test. The cause of Nigerians with BPH and prostatitis having much higher blood PSA levels than Caucasians is yet unknown. The fact that most patients with prostate symptoms in our setting arrive late may be one reason.

PSA values > 20 ng/ml were seen in 60% of the 80 individuals with prostate cancer, and this was associated with rising Gleason scores. Anderson-Jackson *et al.* (2012) and Anunobi *et al.* (2011) reported similar results, observing that PSA levels increased with increasing Gleason scores and were considerably higher in malignant cases. According to Mosli *et al.* (2009), because prostate cancer patients had PSA levels below 4 ng/ml, the PSA threshold for biopsy should be lowered to 2.5 ng/ml. PSA can be detected in different malignancies, but it is a crucial sign for the diagnosis of prostate cancer (Kraus *et al.,* 2010). However, PSA may not be expressed in all cases of prostatic adenocarcinoma. Consequently, despite the widespread use of serum PSA, it is a poor predictor of prostatic tumours. Hence, future diagnostic approaches with higher specificity are warranted.

The most frequent Gleason score in the study we conducted was 7 (33.8%), which indicates moderately differentiated adenocarcinomas. These results are comparable to those of Nwafor et al. (2015) and Albasari *et al.* (2014), who reported proportions of 32.3% and 39.4%, respectively. Gleason scores ≥6 are associated with excellent progression-free survival, according to studies, but scores ≥7 are associated with increased mortality and adenocarcinoma risks, as is the case with many Nigerian patients (Sweat *et al.,* 2002). Raphael et al. (2022) reported that patients with elevated Gleason scores and higher-grade groups have a higher risk of cancer-related complications and mortality in southern Nigeria. Although the Gleason grading system assigns grades to main and secondary patterns to evaluate tumor heterogeneity, subjective evaluation may restrict the method's reliability (Lilleby *et al.,* 2001).

Our study found a statistically insignificant correlation between serum PSA values and Gleason grade groups, which diverges from the findings of Abubakar et al. (2018) and Udoh et al. (2020). This discrepancy highlights the need for further investigation to clarify the relationship between PSA levels and cancer aggressiveness.

The absence of PSA-Gleason correlation suggests that PSA alone may not reliably indicate tumor differentiation in North-western Nigerian men, warranting supplemental biomarkers (e.g., PHI, PCA3) According to Klein et al. (2015), a significant association exists between low socioeconomic status and poorer survival outcomes among prostate cancer patients, even after adjusting for confounding variables. Specifically, men from lower socioeconomic backgrounds face an increased risk of mortality following a prostate cancer diagnosis.

STRENGTHS AND LIMITATIONS OF THE STUDY

Our study is one of the few conducted in this subject area, although it is preliminary and considers the increasing number of prostate cancer patients worldwide. Prostate cancer is becoming more common, especially with elevated PSA levels, according to the current study. This highlights the need for new efforts to combat the disease both within and outside the study region. Our study has limitations; primarily, it only measured serum total PSA, graded the tumor using the Gleason score, which other factors can influence. Therefore, future research should explore additional PSA indices and their relationship with Gleason score, tumor progression, and potential long-term outcomes in northern Nigeria.

CONCLUSION AND RECOMMENDATION

This study demonstrates the high incidence of benign prostatic hyperplasia and prostate cancer, especially in those between the ages of 61 and 70, and the close connection between these conditions with PSA levels. *While PSA correlates with prostatic lesions, its inability to predict Gleason scores highlights the need for enhanced diagnostic protocols in resource-limited settings.*

Our findings highlight the growing burden of prostate cancer in Nigeria, emphasizing the urgent need for enhanced efforts to tackle this health challenge. To improve early detection, we recommend implementing community-based PSA screening with ultrasonography for men aged 50 and above, as well as establishing regional cancer registries.

LIST OF ABBREVIATIONS

BPH:	Benign Prostatic Hyperplasia,
BPH + CP:	Benign Prostatic Hyperplasia and Chronic Prostatitis,
PIN:	prostate intraepithelial neoplasia,
PSA:	prostate-specific antigen,
PCa:	prostate adenocarcinoma
GS:	Gleason score
H and E:	Haematoxylin and Eosin

COMPETING INTEREST

None

FUNDING

None

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

The study was conducted following the principles of the research and ethics committees of Aminu Kano Teaching Hospital (AKTH), Kano, and Ahmadu Bello University Teaching Hospital (ABUTH), Zaria. Ethical approval was obtained from the respective research and ethics committees (AKTH/MAC/SUB/12A/P-3/VI/2837 and ABUTHZ/HREC/W17/2019).

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