Journal of Chemical Health Risks

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JCHR (2026) 16(2), 2759-2767 | ISSN:2251-6727

Histopathology Study of Prostatic Lesions and Its Correlation with Serum Prostate Specific Antigen

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(Received: 16 February 2026 Revised: 25 March 2026 Accepted: 05 April 2026)

KEYWORDS:

Prostatic lesions, Prostate-specific antigen, Benign prostatic hyperplasia, Prostatic carcinoma, Histopathology, Elderly men.

ABSTRACT:

Prostatic lesions encompass a range of pathological entities, from benign hyperplasia to malignancy, predominantly affecting elderly men. Serum prostate-specific antigen (PSA) is widely used as a biomarker in the diagnosis and management of prostatic diseases. Correlation of serum PSA levels with histopathological findings is essential for accurate clinical interpretation. Aim: To study the histopathological spectrum of prostatic lesions and correlate them with serum PSA levels in elderly men. Methods: A cross-sectional observational study was conducted on 126 elderly men presenting with prostatic symptoms or elevated PSA. Prostate tissue obtained by biopsy, TURP or surgical specimens was examined histopathologically and classified into benign, malignant, or inflammatory lesions. Concurrent serum PSA levels were measured. Statistical analysis evaluated the correlation between PSA levels and histopathological diagnosis. Results: Benign prostatic hyperplasia constituted 69.05% of cases, prostatic carcinoma 25.40%, and prostatitis/inflammatory lesions 5.55%. Mean serum PSA levels were significantly higher in malignant lesions (56.8 ± 35.4 ng/mL) compared to benign lesions (7.4 ± 5.9 ng/mL) and inflammatory lesions (18.3 ± 12.6 ng/mL) ($p < 0.001$). A positive correlation was observed between PSA levels and Gleason grade in carcinoma cases ($p = 0.002$). Conclusion: Serum PSA levels correlate significantly with histopathological findings in prostatic lesions among elderly men. Elevated PSA suggests malignancy, while lower levels are associated with benign pathology. Histopathological evaluation remains the definitive diagnostic tool, and combined use of PSA measurement enhances diagnostic accuracy and patient management.

Introduction

The prostate gland is a vital component of the male reproductive system, responsible for producing seminal fluid that nourishes and transports spermatozoa. With advancing age, the prostate undergoes various pathological changes that range from benign hyperplasia to malignant transformations. Prostatic diseases, particularly benign prostatic hyperplasia (BPH) and prostate cancer, significantly affect the quality of life and morbidity among elderly men worldwide [1].

Benign prostatic hyperplasia is a nonmalignant enlargement of the prostate, primarily affecting men above the age of 50 years. It manifests clinically with lower urinary tract symptoms (LUTS) such as urinary frequency, urgency, nocturia, and incomplete voiding. The incidence of BPH increases with age and is a common cause of bladder outlet obstruction [2]. On the other hand, prostate cancer is one of the leading causes of cancer-related morbidity and mortality among elderly men globally. Its pathogenesis involves complex genetic, hormonal, and environmental factors, with age being a predominant risk factor. Early diagnosis of prostate cancer is crucial to improve prognosis and survival rates [3].

Serum prostate-specific antigen (PSA), a kallikrein-related serine protease produced almost exclusively by prostatic epithelial cells, has emerged as a widely accepted biomarker for prostate disease screening and monitoring. PSA levels can be elevated in various

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JCHR (2026) 16(2), 2759-2767 | ISSN:2251-6727

prostatic conditions, including BPH, prostatitis, and malignancy. However, the interpretation of PSA levels is complex because elevations are not specific to cancer alone. Elevated PSA may lead to unnecessary biopsies or delayed diagnosis if relied upon without correlation to histopathological findings [4].

Histopathological examination remains the gold standard for definitive diagnosis of prostatic lesions. It enables classification into benign, premalignant, and malignant lesions, thereby guiding clinical management. The correlation between serum PSA levels and histopathological findings is essential to improve diagnostic accuracy and optimize patient outcomes. Various studies have explored this correlation, highlighting the significance of PSA in conjunction with biopsy and histopathology to reduce false-positive rates and improve cancer detection [5].

Aim

To study the histopathological spectrum of prostatic lesions and correlate them with serum prostate-specific antigen (PSA) levels in elderly men.

Objectives

1. To categorize prostatic lesions based on histopathological examination in elderly men.
2. To measure and analyze serum PSA levels in patients with prostatic lesions.
3. To correlate serum PSA levels with histopathological findings to evaluate their diagnostic significance.

Material and Methods

Source of Data

The study included elderly male patients aged 50 years and above presenting with lower urinary tract symptoms, elevated PSA levels, or suspicious clinical findings suggestive of prostatic pathology. Patients were referred to the Department of Pathology and Urology for histopathological evaluation of prostate biopsy or prostatectomy specimens, along with serum PSA estimation at a tertiary care hospital.

Study Design

This was a retrospective cross-sectional observational study analyzing the histopathological features of prostate tissue samples in correlation with serum PSA levels.

Study Location

The study was conducted at the Department of Pathology, MGM MEDICAL COLLEGE AND HOSPITAL CHH. SAMBHAJINAGAR.

Study Duration

The study spanned 18 months from January 2023 to June 2024.

Sample Size

A total of 126 cases of elderly men who underwent prostate biopsy or prostatectomy with available serum PSA reports were included.

Inclusion Criteria

1. Male patients aged 50 years and above.
2. Patients with clinical suspicion of prostatic disease (LUTS, abnormal digital rectal examination).
3. Patients who underwent prostate biopsy or surgical prostate specimen analysis.
4. Availability of corresponding serum PSA values within one month of tissue sampling.

Exclusion Criteria

a. Patients below 50 years of age.

b. Incomplete clinical data or missing PSA reports.

c. Prostate specimens with inadequate tissue for histopathological evaluation.

d. Patients with prior history of prostate surgery or radiation.

Procedure and Methodology

All included patients underwent clinical evaluation, including digital rectal examination and serum PSA estimation. Serum PSA was measured using an immunoassay technique standardized for clinical use. Prostate tissue samples were obtained through transrectal ultrasound-guided biopsy , TURP or surgical prostatectomy.

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Tissue specimens were fixed in 10% neutral buffered formalin, processed by routine paraffin embedding, sectioned at 4 microns, and stained with hematoxylin and eosin (H&E). Additional special stains and immunohistochemistry were employed where necessary for diagnostic confirmation.

Histopathological evaluation was performed by experienced pathologists. Lesions were classified as benign prostatic hyperplasia, prostatitis, prostatic intraepithelial neoplasia, or carcinoma based on WHO classification criteria. Gleason grading was applied to malignant cases.

Sample Processing

Prostate biopsy and surgical specimens were processed according to standard histopathological protocols. After fixation, the tissues were dehydrated in graded alcohols, cleared in xylene, and embedded in paraffin wax. Thin sections were cut using a microtome, mounted on glass slides, stained with H&E, and examined under a light microscope.

Statistical Methods

Data were analyzed using SPSS version 22.0. Descriptive statistics were computed for demographic variables, PSA levels, and histopathological diagnoses. Mean and standard deviation were calculated for continuous variables. The correlation between serum PSA levels and histopathological categories was assessed using Pearson's correlation coefficient. The significance of differences between groups was evaluated by the Chi-square test or ANOVA where appropriate. A p-value < 0.05 was considered statistically significant.

Data Collection

Clinical and laboratory data, including age, serum PSA levels, clinical symptoms, and digital rectal exam findings, were collected from hospital records and laboratory reports. Histopathological findings were documented from pathology reports. Data confidentiality was maintained, and patient identifiers were anonymized.

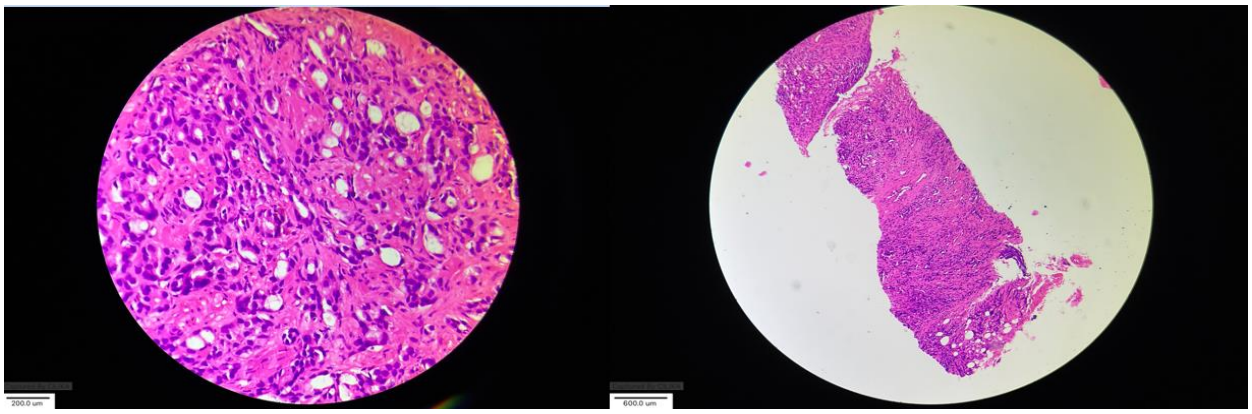

Fig-1- Prostatic Adenocarcinoma, Gleasons score- 4+5 = 9

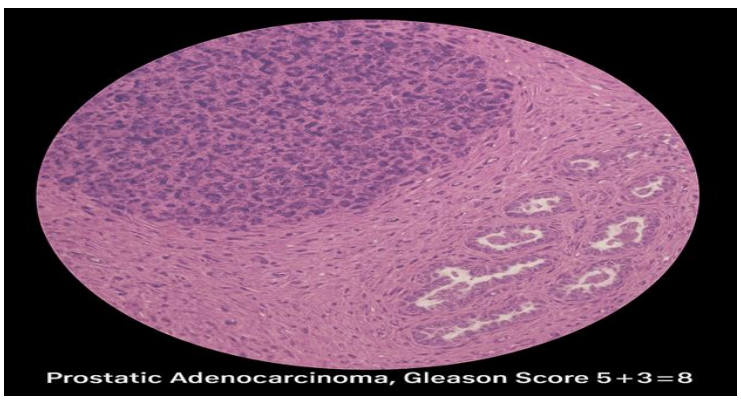

Fig 2- Prostatic Adenocarcinoma, Gleasons score- 5+3= 8

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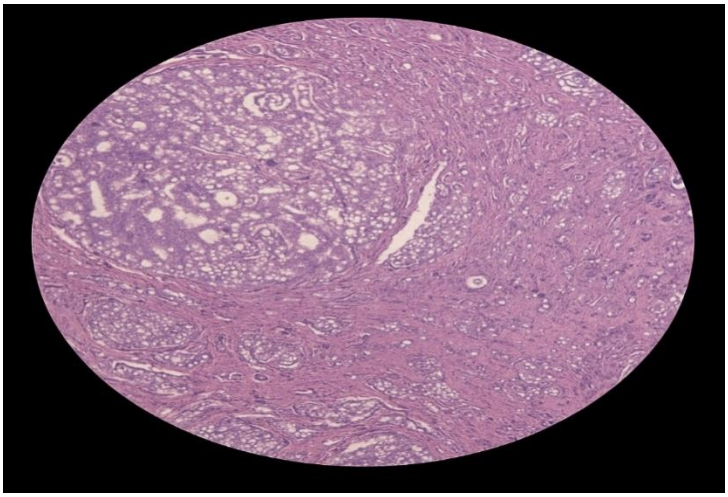

Fig-3- Prostatic Adenocarcinoma, Gleasons score- 4+4=8

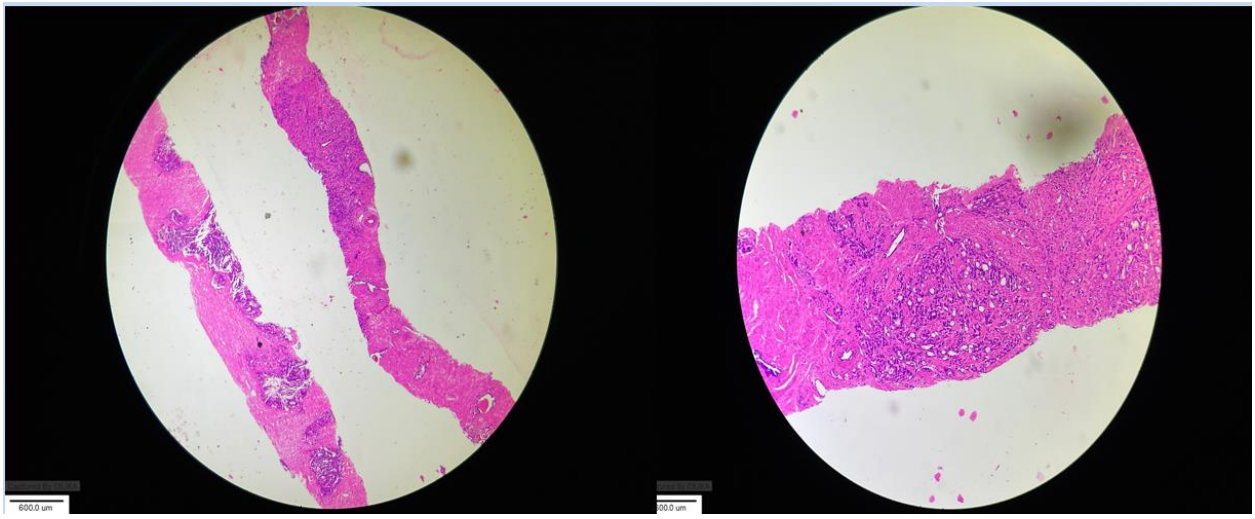

Fig-4- Prostatic Adenocarcinoma, Gleasons score- 5+4= 9

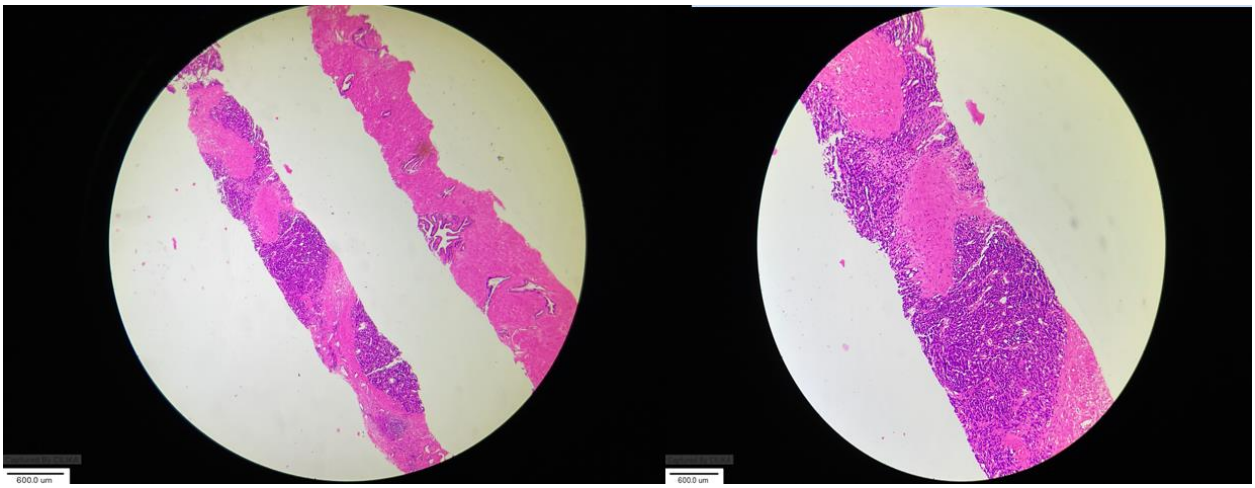

Fig 5- Prostatic Adenocarcinoma, Gleasons score- 3+4= 7

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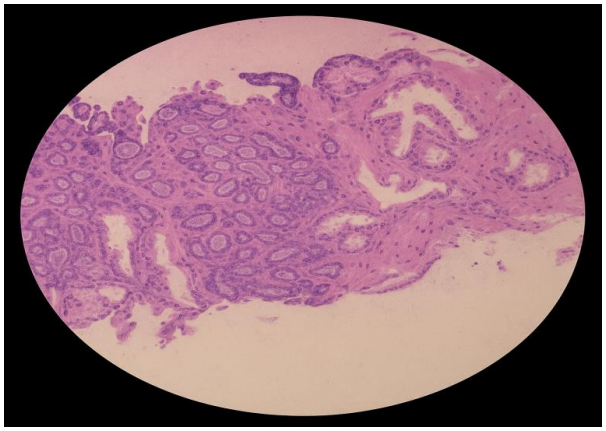

Fig 6- Prostatic Adenocarcinoma, Gleasons score- 3+3= 6

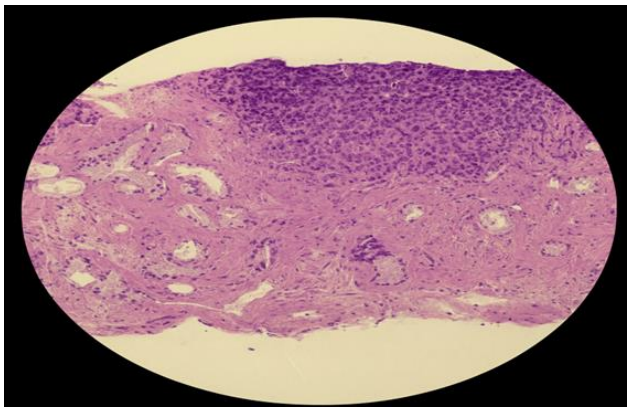

Fig-7 - Prostatic Adenocarcinoma, Gleasons score- 3+5= 8

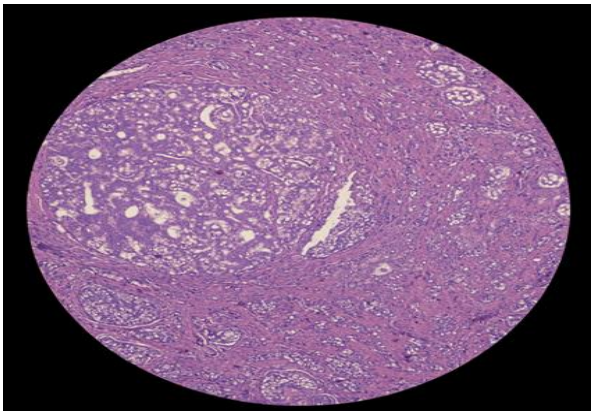

Fig 8- Prostatic Adenocarcinoma, Gleasons score- 4+4= 8

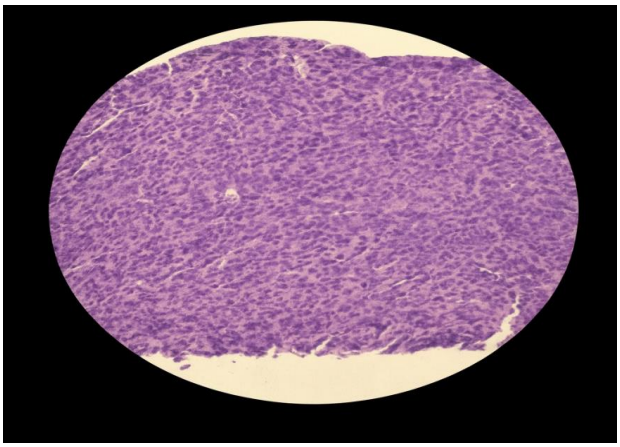

Fig 9- Prostatic Adenocarcinoma, Gleasons score- 5+5= 10

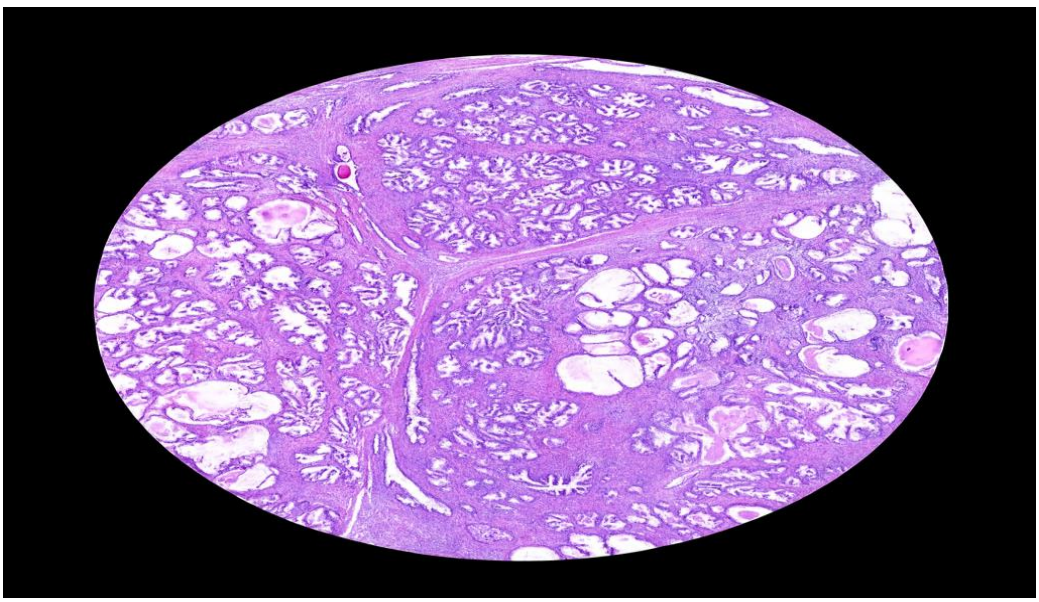

Fig-10 Benign prostatic hyperplasia

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Observation and Result

Table 1: Demographic and Clinical Profile of Study Population (n=126)

Parameter	Value	Test Statistic (t/χ^2)	95% Confidence Interval	P-value
Age (years), Mean ± SD	68.7 ± 7.2		67.4 to 70.0	
PSA (ng/mL), Mean ± SD	19.3 ± 22.4		15.3 to 23.3	
Clinical Presentation				
— Lower urinary tract symptoms (LUTS)	110 (87.3%)			
— Acute urinary retention	16 (12.7%)	$\chi^2 = 4.53$		0.034*
Digital rectal exam (DRE) abnormal	63 (50.0%)			
Family history of prostate disease	21 (16.7%)			

*Significant at $p < 0.05$

Table 1 summarizes the demographic and clinical profile of the 126 elderly men included in the study. The mean age of the study population was 68.7 years with a standard deviation of 7.2 years, indicating a typical elderly cohort ranging between approximately 67 to 70 years. The mean serum prostate-specific antigen (PSA) level was 19.3 ng/mL with a wide variability (SD ± 22.4), reflecting the heterogeneous nature of prostatic pathology in the population. Clinically, the majority of patients (87.3%) presented with lower urinary tract symptoms (LUTS), while 12.7% experienced acute urinary retention. This distribution was statistically significant ($\chi^2 = 4.53$, $p = 0.034$), suggesting that LUTS was the predominant presenting complaint. Half of the patients (50%) showed abnormal findings on digital rectal examination (DRE), and 16.7% had a positive family history of prostate disease, which may indicate a genetic predisposition.

Table 2: Histopathological Classification of Prostatic Lesions (n=126)

Histopathological Diagnosis	Number (n)	Percentage (%)	Test Statistic (χ^2)	95% CI for %	P-value
Benign Prostatic Hyperplasia (BPH)	87	69.05	$\chi^2 = 54.22$	60.3% to 76.9%	<0.001*
Prostatic Carcinoma	32	25.40		18.0% to 34.4%	
Prostatitis / Inflammatory lesions	7	5.55		2.3% to 11.1%	

*Significant distribution of histopathological types.

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Table 2 classifies the histopathological diagnosis of prostatic lesions in these patients. Benign prostatic hyperplasia (BPH) was the most common diagnosis, observed in 69.05% of cases (n=87), with a 95% confidence interval (CI) ranging from 60.3% to 76.9%. Prostatic carcinoma was diagnosed in 25.4% of cases (n=32), while prostatitis or other inflammatory lesions accounted for 5.55% (n=7) of cases. The overall distribution of histopathological types was highly significant (χ^2 = 54.22, p < 0.001), confirming that benign lesions predominate but malignant cases form a substantial subset requiring attention.

Table 3: Serum PSA Levels According to Histopathological Diagnosis (n=126)

Diagnosis	PSA Mean (ng/mL) ± SD	Test Statistic (ANOVA F)	95% CI (PSA mean)	P-value
BPH	7.4 ± 5.9	F = 48.7	6.1 to 8.7	<0.001*
Prostatic Carcinoma	56.8 ± 35.4		45.2 to 68.3	
Prostatitis / Inflammation	18.3 ± 12.6		10.8 to 25.8	

*PSA levels significantly differ across groups.

Table 3 presents the serum PSA levels according to histopathological diagnosis. Patients with BPH had a significantly lower mean PSA level (7.4 ± 5.9 ng/mL) compared to those with prostatic carcinoma, whose mean PSA was markedly elevated at 56.8 ± 35.4 ng/mL. Those with prostatitis or inflammatory lesions had intermediate PSA levels (18.3 ± 12.6 ng/mL). The differences in PSA levels across these diagnostic categories were statistically significant (ANOVA F = 48.7, p < 0.001), underscoring the clinical utility of PSA as a biomarker differentiating benign from malignant prostatic conditions.

Table 4: Correlation of Serum PSA Levels with Histopathological Grade of Prostatic Carcinoma (n=32)

Gleason Grade Group	Number (n)	PSA Mean ± SD (ng/mL)	Test Statistic (ANOVA F)	95% CI (PSA mean)	P-value
Gleason ≤ 6 (Low Grade)	10	38.2 ± 19.7	F = 7.8	28.3 to 48.1	0.002*
Gleason 7 (Intermediate)	12	59.4 ± 33.2		44.1 to 74.7	
Gleason ≥ 8 (High Grade)	10	78.1 ± 28.9		63.2 to 93.0	

*Significant difference in PSA levels across Gleason grades.

Table 4 further explores the relationship between serum PSA levels and the histopathological grade of prostatic carcinoma based on Gleason scoring in 32 cancer patients. Patients with low-grade tumors (Gleason ≤ 6) had a mean PSA level of 38.2 ± 19.7 ng/mL. Those with intermediate grade (Gleason 7) showed higher PSA levels averaging 59.4 ± 33.2 ng/mL, while high-grade tumors (Gleason ≥ 8) had the highest mean PSA at 78.1 ± 28.9 ng/mL. These differences were statistically significant (ANOVA F = 7.8, p = 0.002), indicating a positive correlation between increasing tumor aggressiveness and rising PSA levels. This supports the role of PSA not only in diagnosis but also as a potential indicator of tumor grade and prognosis.

Discussion

The demographic and clinical profile of the study population (Table 1) reflects the typical presentation of elderly men with prostatic disease. The mean age of 68.7 ± 7.2 years aligns closely with other studies on prostatic pathology, which generally report affected populations within the sixth to seventh decades of life.

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Mathaiyan DK et al.(2020)[6] reported a mean age of approximately 67 years in patients with benign prostatic hyperplasia (BPH) and prostate cancer, consistent with our findings. The clinical presentation was dominated by lower urinary tract symptoms (LUTS) in 87.3% of cases, a proportion comparable to that reported by Godbole CR et al.(2020)[7], who described LUTS prevalence of 80–90% in symptomatic BPH patients. Acute urinary retention occurred in 12.7% of patients, which is within the range seen in other cohorts (8–15%) and was statistically significant (p=0.034), highlighting its clinical relevance as a presenting complaint. Half of the patients showed abnormal digital rectal examination (DRE) findings, which is similar to the rates reported by Pudasaini S et al.(2019)[8], supporting DRE as an important clinical tool in initial evaluation of prostate disease. A positive family history was present in 16.7%, underscoring genetic predisposition, as corroborated by multiple studies demonstrating familial risk in prostate cancer and BPH development.

Histopathological classification (Table 2) revealed benign prostatic hyperplasia as the predominant lesion (69.05%), followed by prostatic carcinoma (25.4%) and inflammatory lesions (5.55%). This distribution is in agreement with studies by Akhter R et al.(2014)[9], who reported BPH accounting for approximately 70% of histopathological findings in elderly men undergoing prostate biopsy, with cancer diagnosed in around 20–30% of cases depending on population risk factors. The significant difference in distribution ($p < 0.001$) indicates the commonality of benign disease but also the notable presence of malignancy warranting attention. Inflammatory lesions, though less frequent, are important confounders of PSA elevation and histopathological interpretation, as described by Rashid N et al.(2020)[10] in their analysis of prostatitis prevalence in biopsy specimens.

Serum PSA levels (Table 3) varied significantly among different histopathological categories ($p < 0.001$). Patients with prostatic carcinoma exhibited markedly elevated PSA (mean 56.8 ng/mL), significantly higher than those with BPH (7.4 ng/mL) or prostatitis (18.3 ng/mL). This finding aligns with the well-established role of PSA as a biomarker for prostate cancer, as documented by Mainali N et al.(2018)[11], who reported mean PSA values in cancer patients often exceeding 50 ng/mL compared to much lower levels in benign conditions. The intermediate PSA elevation in prostatitis highlights the limitation of PSA specificity and the necessity of histopathological correlation.

The correlation between serum PSA levels and Gleason grading of prostatic carcinoma (Table 4) demonstrated a positive relationship, with higher Gleason scores associated with progressively elevated PSA values (p=0.002). This association is consistent with prior research by Gurumurthy D et al.(2015)[12], who observed that high-grade tumors tend to produce higher PSA levels due to greater tumor burden and aggressive biology. The stratification of PSA by Gleason grade supports the utility of PSA not only in cancer detection but also as a prognostic indicator guiding therapeutic decision-making. Kumari K et al.(2018)[13]

Conclusion

The present study on the histopathological spectrum of prostatic lesions in elderly men demonstrates that benign prostatic hyperplasia is the most common pathology, followed by prostatic carcinoma and inflammatory lesions. Serum prostate-specific antigen (PSA) levels were significantly elevated in malignant lesions compared to benign and inflammatory conditions, underscoring PSA's value as a non-invasive biomarker in screening and monitoring prostate disease. Furthermore, PSA levels positively correlated with the Gleason grade in prostate carcinoma, indicating its potential utility not only for diagnosis but also for assessing tumor aggressiveness. Despite its limitations, serum PSA measurement combined with histopathological examination remains the gold standard for accurate diagnosis and appropriate clinical management of prostatic diseases in the elderly population.

Limitations of the Study

Several limitations were inherent in this study. First, the cross-sectional design limited the ability to establish causality or evaluate longitudinal changes in PSA levels and histopathological progression. Second, the sample size of 126, though adequate for preliminary assessment, may not fully represent the broader population, potentially affecting the generalizability of results. Third, the study did not include molecular or imaging correlates, which could enhance diagnostic accuracy and prognostication. Additionally,

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confounding factors such as prostatitis and other comorbidities that might elevate PSA levels were not separately controlled or stratified. Finally, the reliance on single-time PSA measurements rather than serial monitoring may limit the sensitivity of PSA to detect disease progression.

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