Section: Pathology

Original Research Article

CLINICO-PATHOLOGICAL STUDY OF TRUS GUIDED NEEDLE BIOPSY OF PROSTATE IN PATIENTS HAVING HIGH SERUM PSA LEVEL WITH SPECIAL REFERENCE TO IMMUNO-HISTOCHEMISTRY

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ABSTRACT

Background: Prostate cancer is one of the most common malignancies in men worldwide. It is the leading non-skin malignancy in men and the second-leading cause of cancer death after lung cancer. Early detection relies on prostate-specific antigen (PSA) screening and imaging-guided biopsy. However, PSA has limited specificity as it can be elevated in benign conditions. Transrectal ultrasonography (TRUS)–guided core biopsy is the gold standard for diagnosis. Immunohistochemical (IHC) markers such as high-molecular-weight cytokeratin (34βE12) and AMACR (α-methylacyl-CoA racemase, P504S) are increasingly used to distinguish benign from malignant prostate lesions. This study evaluates the clinicopathological features of TRUS-guided prostate biopsy in patients with high PSA and the role of IHC in diagnostic accuracy.

Materials and Methods: We conducted a hospital-based cross-sectional study of patients (age ≥50) with lower urinary tract symptoms (LUTS), elevated PSA, and/or abnormal digital rectal examination (DRE) undergoing TRUS-guided prostate biopsy. A total of 45 cases were collected (May 2020–April 2021) at North Bengal Medical College, Darjeeling. Biopsy specimens were processed with routine hematoxylin–eosin staining. Cases with atypical or suspicious foci were further evaluated by IHC using antibodies against 34βE12 (basal cell marker) and AMACR (cancer marker). Data were analyzed descriptively; categorical comparisons used chi-square or Fisher's exact test ($p<0.05$ significant).

Results: Forty-two biopsy cases were evaluable after excluding 3 nondiagnostic cores. The mean patient age was 65.6±6.5 years. Patients aged 60–69 years comprised the majority (57.1%) (Table 1). Most patients presented from rural areas (64.3%). Common symptoms were increased urinary frequency and nocturia (each 59.5%). On DRE, 47.6% had a hard nodule, while 35.7% had an enlarged firm prostate. On TRUS, 35.8% had an enlarged gland without focal lesion, 33.3% had a focal nodule, and 30.9% had multifocal nodules. Serum PSA levels were most commonly between 10.1–20 ng/mL (40.5%), and the proportion of adenocarcinoma increased with rising PSA levels, accounting for 70.5% of cases with PSA 10.1–20 ng/mL and 80.0% of those with PSA >20 ng/mL. Histopathology showed 43.0% adenocarcinoma, 35.7% nodular hyperplasia, 14.2% prostatic intraepithelial neoplasia (PIN), and 7.1% carcinoma mimickers (Table 4). Among 18 adenocarcinomas, Gleason scores were distributed across all grade groups (Group 1–5). IHC confirmed malignancy in equivocal cases. AMACR was positive in 17/18 (94.4%) carcinoma foci, with one false-negative (5.6%). All benign mimickers and hyperplasia were negative for AMACR. Basal cell

marker 34βE12 highlighted intact basal layers in benign and PIN lesions, but was absent in carcinoma foci.
Conclusion: In this cohort of high-PSA patients, nearly half had carcinoma on biopsy. The use of dual IHC (AMACR and 34βE12) improved diagnostic confidence, with high sensitivity (94.4%) for detecting cancer foci. These findings reinforce the importance of systematic 12-core TRUS biopsy and adjunct IHC markers in accurately diagnosing prostate cancer.
Keywords: Prostate biopsy; TRUS-guided; PSA; AMACR; basal cell marker; prostate adenocarcinoma.

INTRODUCTION

Prostate cancer is a major health concern for men globally. It is the most commonly diagnosed cancer in men (aside from non-melanoma skin cancers) and the second leading cause of cancer mortality after lung cancer.[1,2] In India and worldwide, incidence rises with age; most patients are over 65 years old. Screening with prostate-specific antigen (PSA) and digital rectal examination (DRE) has increased early detection, but elevated PSA is not specific.[3,4,5] Benign prostatic hyperplasia (BPH), prostatitis, and other conditions can raise PSA, leading to false positives.[3,4] For example, studies show up to 86% of BPH patients can have elevated PSA levels despite no cancer. In clinical practice, a PSA >4.0 ng/mL is typically used as a threshold; at this cut-off the test has a sensitivity of ~91% for cancer, though specificity is much lower. DRE alone is even less sensitive or specific.

Given these limitations, prostate biopsy remains the definitive diagnostic step.[6,7] Since Hodge et al,[7] first described ultrasound-guided biopsy, systematic sampling (initially sextant and later extended 12-core protocols) has been standard.[8] Increasing the number of cores (e.g. from 6 to 12) improves cancer detection rates without significant harm.[8,9] In fact, extended 12-core biopsies can improve the detection rate by ~20% compared to sextant sampling. The 12-core TRUS-guided biopsy is now standard practice for men with abnormal PSA or DRE.

Pathological examination of biopsy cores by H&E staining usually suffices, but small foci of cancer can be difficult to distinguish from benign mimickers (atrophy, adenosis, PIN) based on morphology alone.[12,13,15] To augment accuracy, immunohistochemical markers are used. The basal cell markers (high-molecular-weight cytokeratin 34βE12 and p63) highlight benign glands by staining basal cells, which are absent in adenocarcinoma. Conversely, the enzyme AMACR (alpha-methylacyl-CoA racemase, also known as P504S) is overexpressed in carcinoma cells but not in benign glands. Dual staining for AMACR (positive in cancer) and 34βE12 or p63 (negative in cancer) is a powerful "cocktail" that increases diagnostic sensitivity and specificity. For example, AMACR IHC has shown ~97% sensitivity and 100% specificity for prostate cancer in needle biopsies.[10,11,14]

In this study, we performed a clinico-pathological analysis of 42 TRUS-guided prostate biopsies from men with high PSA. We assessed clinical presentation, DRE/TRUS findings, and histopathology. We also evaluated the diagnostic contribution of IHC with AMACR and 34βE12 in cases with suspicious or ambiguous histology.

MATERIALS AND METHODS

A cross-sectional study was conducted in the Departments of Pathology, Urology, and Radiodiagnosis at North Bengal Medical College (May 2020–April 2021). Patients were recruited by consecutive sampling. Inclusion criteria were: age ≥50 years, presentation with LUTS and/or abnormal DRE, and elevated serum PSA. Exclusion criteria included history of known prostate cancer, prostatitis, urinary tract infection, or recent urologic instrumentation. Written informed consent was obtained.

Each patient underwent transrectal ultrasonography (TRUS) and 12-core systematic prostatic biopsy under antibiotic prophylaxis. Biopsy cores were fixed in formalin, paraffin-embedded, and sectioned. Slides were stained with hematoxylin–eosin for routine histopathological diagnosis. When the H&E findings were equivocal or suspicious (foci of atypia, ASAP, HGPIN), additional immunohistochemical staining was performed. A dual staining protocol was used: cocktail of 34βE12 (high-molecular-weight cytokeratin clone 34βE12, basal cell marker) and AMACR (clone 13H4, cancer specific marker) according to standard two-step indirect method.

Data collected included patient age, residence, presenting symptoms, PSA level, DRE and TRUS findings. Histopathological categories were recorded: benign nodular hyperplasia, adenocarcinoma, PIN (low/high-grade), or benign mimickers. The Gleason score/grade group was noted for carcinomas. AMACR and 34βE12 staining results were interpreted as positive (cytoplasmic granular staining for AMACR; cytoplasmic/membranous for 34βE12) or negative. AMACR grading was done. Final diagnosis incorporated morphology and IHC results.

Statistical analysis used SPSS. Descriptive statistics (mean±SD, counts, percentages) were calculated. Categorical data were compared using chi-square or

Fisher's exact test. Significance threshold was $p<0.05$.

RESULTS

A total of 45 biopsies were performed. Three cases were excluded (two with inadequate tissue, one nondiagnostic). Final analysis included 42 patients (Table 1). The mean age was 65.6±6.5 years. Table 1 shows the age distribution. The majority were aged 60–69 years (57.1%). Serum PSA levels ranged from <4 to >20 ng/mL, with most patients having PSA levels between 10.1–20 ng/mL (40.5%). Higher PSA levels were associated with an increased frequency of adenocarcinoma, accounting for 70.5% of cases with PSA 10.1–20 ng/mL and 80.0% of those with PSA >20 ng/mL (Table 3). Most patients (64.3%) were from rural areas (Table 3). Common symptoms included increased urinary frequency and nocturia (each in 25/42 patients, 59.5%); urinary hesitancy, urgency, poor stream & hematuria also occurred.

Table 1: Age distribution of participants (n=42)

Age Group (years)	Frequency	Percentage (%)
50–59	6	14.3%
60–69	24	57.1%
≥70	12	28.6%
Total	42	100.0%

Table 2: Residence of participants (n=42). Majority lived in rural areas

Place of Residence	Frequency	Percentage (%)
Rural	27	64.3%
Urban	15	35.7%
Total	42	100.0%

Table 3: Distribution of study participants according to Total serum PSA level (ng/ml) and histopathological diagnosis

Serum PSA Level (ng/mL)	NHP n (%)	Cancer Mimickers n (%)	PIN n (%)	Adenocarcinoma n (%)	Total, n (%)
≤4	1 (25.0)	1 (25.0)	1 (25.0)	1 (25.0)	4 (9.5)
4.1–10	11 (68.8)	1 (6.2)	3 (18.8)	1 (6.2)	16 (38.1)
10.1–20	2 (11.8)	1 (5.9)	2 (11.8)	12 (70.5)	17 (40.5)
>20	1 (20.0)	0 (0.0)	0 (0.0)	4 (80.0)	5 (11.9)
Total	**15 (35.7)**	**3 (7.1)**	**6 (14.3)**	**18 (42.9)**	**42 (100.0)**

On examination, DRE findings were as follows: 20 patients (47.6%) had a hard prostatic nodule, 15 (35.7%) had a diffusely enlarged firm gland, 6 (14.3%) had a suspicious (focal) nodule, and 1 (2.4%) had an enlarged nodular gland. Fig. 2 illustrates these DRE findings. On TRUS, 15 (35.8%) had a homogeneously enlarged prostate, 14 (33.3%) had a single focal lesion, and 13 (30.9%) had multifocal lesions.

Table 4: Histopathological findings on TRUS biopsy (n=42). Adenocarcinoma was the most common diagnosis.

Diagnosis on H&E	Frequency	Percentage (%)
Nodular hyperplasia (benign)	15	35.7%
Carcinoma mimickers (benign variants)	3	7.1%
Prostatic intraepithelial neoplasia	6	14.2%
Adenocarcinoma	18	43.0%
Total	42	100.0%

Table 4 indicates that 18/42 patients (43.0%) had prostate adenocarcinoma. Among the 18 carcinomas, Gleason grade groups were distributed across groups 1–5 (Table 5). Two cases were grade group 1 (Gleason score ≤6), three grade group 2 (3+4), seven grade group 3 (4+3), two grade group 4 (Gleason score 8), and four grade group 5 (Gleason score 9–10).

Table 5: Gleason score/Grade group distribution in 18 prostate adenocarcinomas.

Gleason Score	Grade Group	Frequency	Percentage (%)
≤6 (3+3)	Group 1	2	11.1%
3+4	Group 2	3	16.7%
4+3	Group 3	7	38.9%
8 (4+4)	Group 4	2	11.1%
9–10	Group 5	4	22.2%
Total		18	100.0%

Combined interpretation of HPE and IHC yielded the final diagnoses listed in Table 6. Table 6 compares diagnoses by routine HPE vs IHC vs final consensus. In our study, on histopathological examination, out of 42 cases, 18 were diagnosed as prostatic adenocarcinoma, 15 cases as nodular

hyperplasia (including one case of NHP with chronic prostatitis), 4 cases as HGPIN, 2 cases as LGPIN, 2 cases of prostatic atrophy & one case of basal cell hyperplasia. Out of 18 prostatic carcinoma cases, 1 case of Mixed Acinar & Ductal Adenocarcinoma was identified, rest 17 cases were found to be Acinar Adenocarcinoma on HPE.

On histopathology, 3 cases were found to be difficult to diagnose & report. One case was diagnosed as prostatic atrophy & reported with a suggestion of immunohistochemistry to rule out malignancy which was further diagnosed & confirmed as a case of prostatic adenocarcinoma on subsequent immunohistochemistry with AMACR & 34βE12. Another histopathologically reported case; suspicious of prostatic adenocarcinoma (atrophic variant) was ruled out by immunohistochemistry & a final diagnosis of prostatic atrophy was made. Another case which was reported as suspicious foci of prostatic adenocarcinoma on routine HPE with a suggestion of IHC evaluation for confirmation of malignancy was found to be a case of Basal Cell Hyperplasia on further immunohistochemistry.

On immunohistochemistry, one case of NHP showed false negative staining with HMWCK & one case of LGPIN showed false negative AMACR expression. Out of 18 adenocarcinoma cases, 15 cases showed AMACR positivity & HMWCK negativity in the malignant foci. 1 case of adenocarcinoma showed negative staining with both AMACR (false negative) & HMWCK & another 2 cases of prostatic adenocarcinoma showed positive staining result with dual cocktail antibody giving rise to false positive expression with HMWCK.

So, in this study, the final diagnosis was achieved by combining routine histopathology & immunohistochemistry as per our study protocol. However, to achieve final diagnosis in Prostatic Adenocarcinoma, a combined approach of routine histopathology & immunohistochemistry was found to be more effective by ruling out benign mimickers & ruling in malignant lesions mimicking benign glands.

As per final diagnosis, out of 42 cases, 18 were prostatic carcinoma, 15 were nodular hyperplasia, 4 cases were HGPIN, 2 cases were LGPIN, 2 cases were prostatic atrophy & 1 case was basal cell hyperplasia.

Table 6: Final diagnosis reconciliation. IHC (AMACR/34βE12) resolved 3 cases that routine histology had missed.

Lesion Type	Routine Diagnosis on HPE	Diagnosis by IHC	Final Diagnosis
Nodular hyperplasia	15	14	15
Basal cell Hyperplasia	1	2	2
Prostatic Atrophy (PA)	2	1	1
LGPIN	2	1	2
HGPIN	4	4	4
Adenocarcinoma	18	15	18

Table 6: Comparisons of diagnoses by routine H&E, by IHC staining, and final integrated diagnosis (n=42).

AMACR was positive in 17/18 (94.4%) of confirmed carcinoma cases and negative in benign mimickers. Specifically, AMACR showed moderate to strong cytoplasmic staining in most adenocarcinoma foci. One carcinoma case (Gleason 4+3) lacked AMACR expression (false negative). All 15 nodular hyperplasias and benign mimics were AMACR-negative. The sensitivity of AMACR for detecting carcinoma in this series was thus 94.4% with 79.1% specificity. AMACR grading (grade 0 to 3) was done depending upon the intensity of cytoplasmic staining patterns. 12/18 (66.7%) cases of prostatic adenocarcinoma showed grade 3 (strong) AMACR positivity (Figure 3).

34βE12/ HMWCK staining highlighted intact basal cells in 14/15 NHP cases (one case showed false negative result) & in all the benign mimickers and was lost in 16 out of 18 carcinoma cases. The sensitivity of HMWCK in diagnosing prostatic adenocarcinoma was thus 88.8% with 91.6% specificity. All the 4 HGPIN cases showed discontinuous positivity for basal cells. Out of 2 LGPIN cases, one case was HMWCK negative & one case showed discontinuous positivity for basal cells.

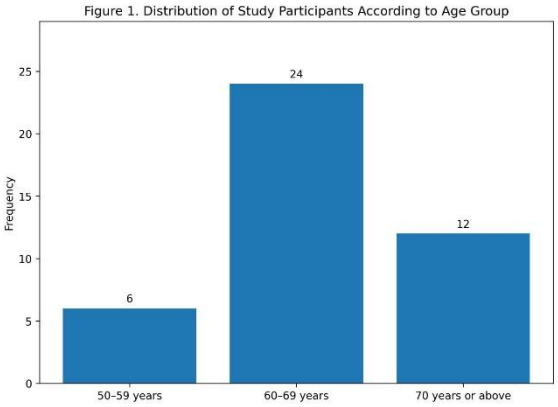

Figure 1: Age distribution of study participants

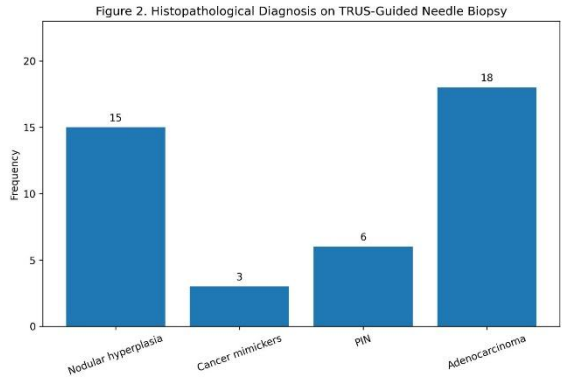

Figure 2: Histopathological diagnosis on TRUS-guided prostate biopsy

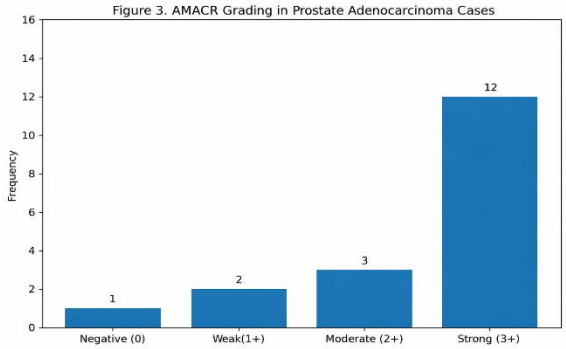

Figure 3: AMACR expression grading in prostate adenocarcinoma

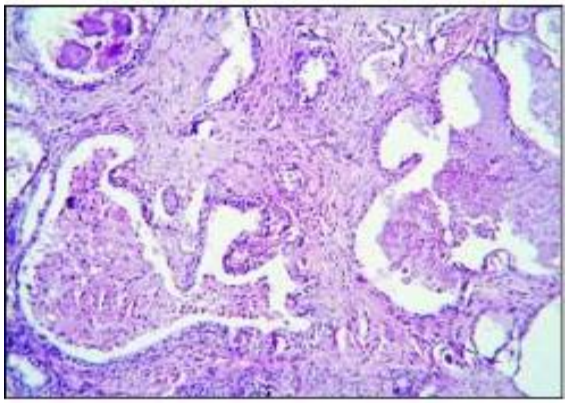

Figure S1: Benign prostate glands with intraluminal secretions in a case of NHP; H&E Stain; (10 X)

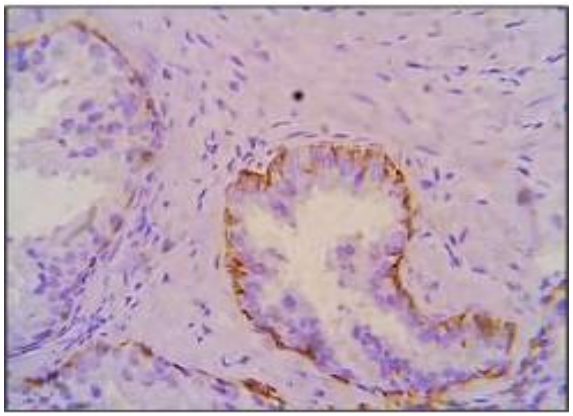

Figure S2: Continuous 34βE12 positivity of basal cell layer in Benign Prostatic Glands; (40X).

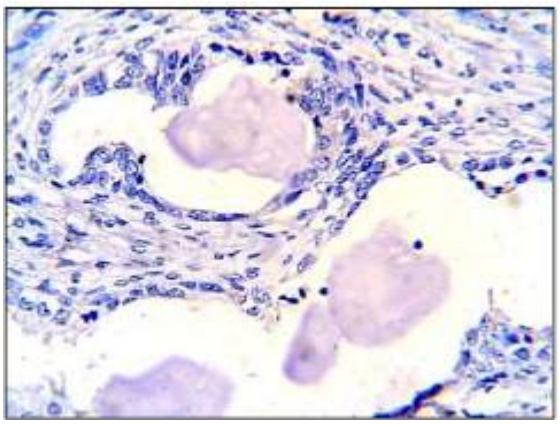

Figure S4: Atrophic Glands; H&E Stain; (10X)

Figure S5: Continuous 34βE12 Positivity in Prostatic Atrophy (10X)

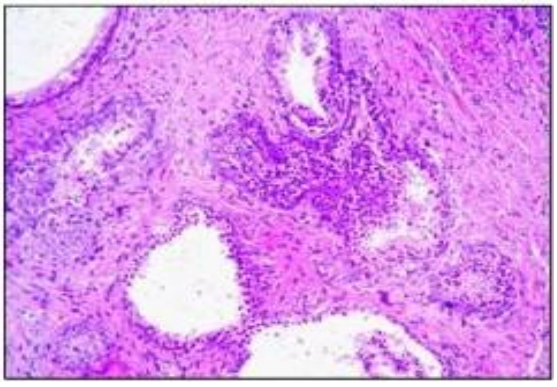

Figure S6: Basal cell hyperplasia; H&E (40X)

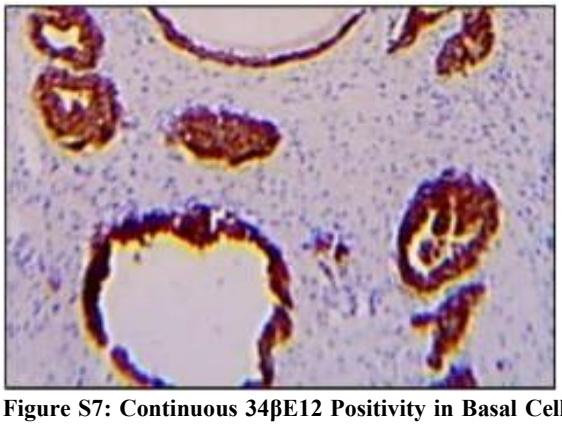

Figure S7: Continuous 34βE12 Positivity in Basal Cell Hyperplasia shows multilayering of basal cells (40X).

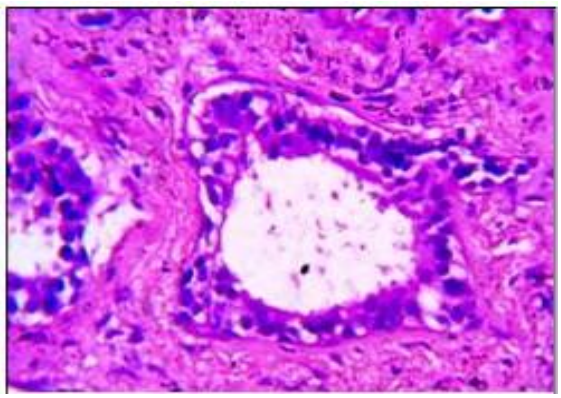

Figure S8: Low Grade Prostatic Intraepithelial Neoplasia; H&E (40X).

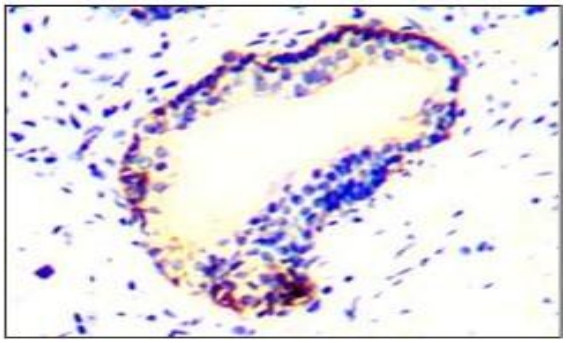

Figure S9: Discontinuous 34βE12 positivity of basal cell layer in LGPIN (40X).

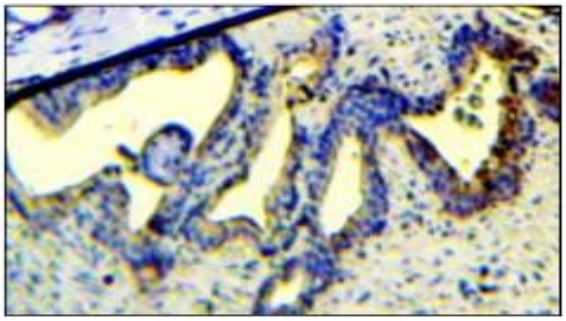

Figure S10: Weak & Non-circumferential Positivity of AMACR in luminal epithelial Cells in PIN (40X).

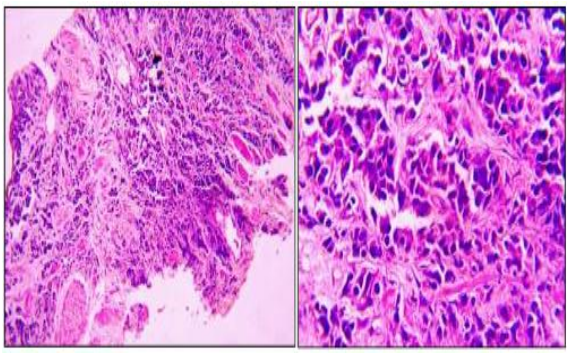

Figure S11: Predominantly fused glands, cords & islands of malignant cells with very few well formed glands in Prostate Adenocarcinoma (Gleason's Score 4+3=7), H&E, 10X (Left) & 40X (Right).

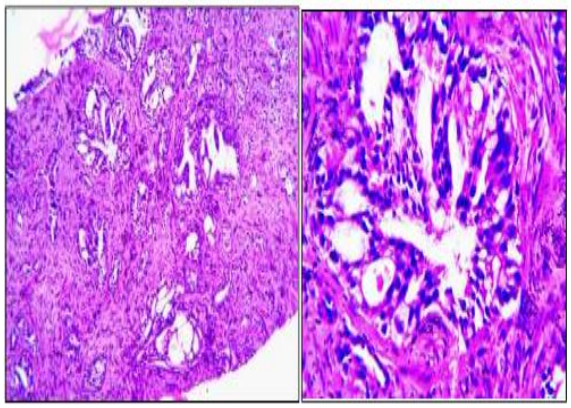

Figure S12: Prostatic Acinar Adenocarcinoma, Gleason's Score 5+4=9:H&E, 10X (Left) & 40X (Right) showing fused glands with slit like spaces (CRIBRIFORM PATTERN).

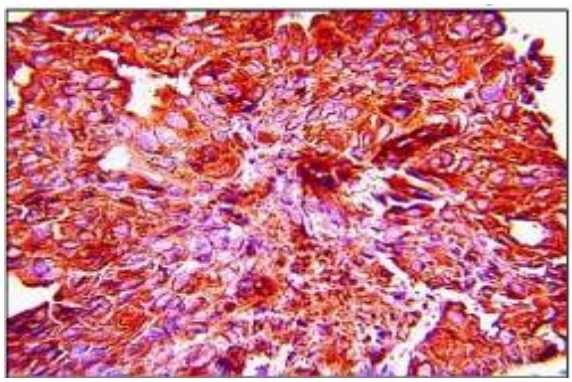

Figure S13: AMACR shows dark diffuse, strong & granular cytoplasmic positivity (GRADE 3) of malignant foci in Adenocarcinoma Prostate in needle biopsy, H&E (40X).

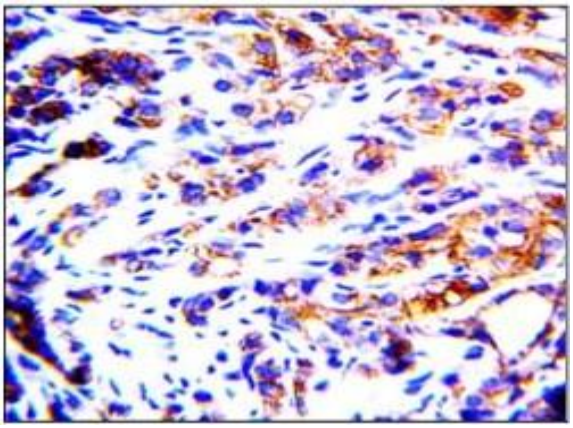

Figure S14: AMACR shows diffuse, moderate, granular cytoplasmic positivity (GRADE 2) in Prostatic Adenocarcinoma (10X).

DISCUSSION

Our study demonstrated that a high proportion (43%) of men with elevated PSA and/or abnormal DRE had biopsy-proven prostate cancer.[1,2,8,9] This detection rate is comparable to other reports using 12-core TRUS biopsy in symptomatic patients. We found that most cancers occurred in men in their 60s (mean age 65.6 years), consistent with

epidemiologic data that risk rises after 50.[1,2,4] Most participants presented with lower urinary tract symptoms (frequency, nocturia) rather than hematuria. Digital rectal exam was useful: nearly half had a discrete hard nodule, and only those with cancer had hematuria. These findings align with known clinical patterns – cancer can be asymptomatic or mimic benign enlargement until advanced.

The TRUS biopsy procedure (12 systematic cores) yielded a mix of benign and malignant diagnoses. The nodular hyperplasia seen in 36% of cases reflects the common finding of BPH in this age group.[3,4] Adenocarcinoma constituted the plurality (43%). Some older series in India report detection rates around 25–40% in similar patient groups; our higher rate may be due to stricter inclusion (all had high PSA) and thorough IHC confirmation. As in many studies, raising the number of biopsy cores improves yield.[8,9] Our results support that approach.

The Gleason grade distribution was broad; however, most cancers were intermediate-grade (Grade groups 2–3). Notably, even low-grade lesions (Gleason ≤6) were detected. This underscores that systematic biopsy can find early cancers that might benefit from surveillance or early treatment.

The key aspect of our work was the use of adjunct IHC. Prostate biopsies often contain small atypical foci where H&E alone is equivocal. In such cases, an IHC panel significantly enhances accuracy. We found that AMACR was highly sensitive (17/18 positive) for cancer foci, echoing prior studies (e.g. Zhou et al,[11] reported 97% sensitivity, 100% specificity).[10,14] 34βE12 (a basal cell cytokeratin) confirmed benign gland architecture when present. By combining AMACR (positive in cancer) with 34βE12 (negative in cancer), we resolved all ambiguous cases. The triple cocktail has been recommended by the International Society of Urologic Pathology (ISUP) for diagnostically challenging biopsies.[15] One cancer case (Gleason 4+3) was negative for AMACR (5.6% false-negative), a known limitation described by others. Conversely, 34βE12 occasionally gave false positives if benign mimickers lacked basal cells. Hence, interpretation must consider both stains together.

Our findings have practical implications: routine use of a 12-core TRUS biopsy combined with AMACR/34βE12 IHC can maximize cancer detection while minimizing misdiagnosis[6, 8, 9, 15]. Given that PSA screening can produce many false positives, histopathological confirmation is crucial. The high sensitivity of AMACR means a positive stain strongly favors cancer, whereas basal markers help avoid overcalling benign lesions. Similar clinicopathological studies have endorsed this IHC approach as reliable for small foci.

Limitations of this study include its single-center design and relatively small sample. We did not include MRI-guided biopsies, which can increase detection further. Future work could compare systematic vs. MRI-targeted biopsies in this population. Nonetheless, our data confirm existing knowledge: most prostate cancers occur in older men with high PSA, and histological confirmation aided by IHC is necessary for accurate diagnosis.

CONCLUSION

TRUS-guided core biopsy remains essential for diagnosing prostate cancer in high-PSA patients. In this series, 43% of biopsies were malignant, predominantly in men aged 60–69, majority belonged to rural areas. We concluded that serum PSA value was a useful tool in stratifying the patients & guiding for appropriate selection of patient for further evaluation. TRUS was extremely valuable tool in knowing the nature of lesion and making a primary impression of type of lesion along with procurement of adequate sample for proceeding with HPE examination. Routine HPE still remain the primary examination in detecting the malignancy in case of prostate adenocarcinoma. HPE was sufficient to reach diagnosis of prostatic adenocarcinoma or NHP yet in some cases we found it difficult to reach the diagnosis such as in case of cancer mimickers, PIN, and others. Routine HPE still remain the primary examination in detecting the malignancy in case of prostate adenocarcinoma. HPE was sufficient to reach diagnosis of prostatic adenocarcinoma or NHP yet in some cases we found it difficult to reach the diagnosis such as in case of cancer mimickers, PIN, and others. In such suspicious lesion IHC played its valuable role in making of proper diagnosis. It is better to use a panel of IHC with one being a positive marker and at least one being a negative marker for adenocarcinoma. In our study we were able to observe and compare the sensitivity & specificity of the markers and found to be good tool for reaching diagnosis of various prostatic lesions.

Limitations

1. In our study, the number of study participants were only 42. More number of cases & a longer duration of the study was needed to provide a more accurate & comprehensive analysis.
2. Obtaining a greater number of biopsy cores & subsequent histopathological examination would have been improve the diagnostic accuracy.
3. Inclusion of serum free PSA & other indices (e.g., PSA velocity) in the study could have been helpful in distinguishing prostate cancer from benign disease in men with a borderline total PSA range (between 4 to 10 ng/ml).
4. Availability of IHC Tripple Cocktail could have shown more fruitful results.

Consent: Obtained

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