Section: Pathology

Original Research Article

CORRELATION OF IMMUNOHISTOCHEMICAL EXPRESSION OF PROSTATE SPECIFIC MEMBRANE ANTIGEN (PSMA) AND GLYOXALASE -1 WITH SERUM PSA ACROSS THE HISTOMORPHOLOGICAL SPECTRUM OF PROSTATIC LESIONS

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ABSTRACT

Background: Lesions responsible for prostatic morbidity and mortality among men vary across spectrum of histomorphological entities including benign nodular hyperplasia, prostatic carcinomas and their precursors. PSMA has been found in the prostatic secretory cells while Glyoxalase -1 upregulation has been observed in states of high metabolic and inflammatory stress particularly in cancers. In current study we aim to study the expression of PSMA and Glyoxalase -1 across this spectrum of prostatic lesions in relation with serum PSA.

Materials and Methods: Present study included 60 prostatic specimens comprising of 48 TURP chips, 11 prostatic biopsies and 1 prostatectomy specimen. Histopathological results were grouped into non-neoplastic and neoplastic lesions. PSMA and Glyoxalase -1 expression were correlated with Gleason Score and Gleason Grade Group in cases of prostatic adenocarcinoma.

Results: Out of 60 prostatic specimens from patients with mean age of 68.03 years and mean serum PSA 13.31ng/mL, majority of the prostatic lesions were benign (46 cases) followed by neoplastic lesions including prostatic carcinomas (13 cases) and 1 case of Prostatic intraepithelial neoplasia. PSMA and Glyoxalase -1 had weak to moderate cytoplasmic/membranous and weak to moderate cytoplasmic expression respectively in the benign cases along with direct proportionality of PSMA expression with Gleason score and grade group while Glyoxalase-1 expression had considerable variation.

Conclusion: Varied expression of PSMA and Glyoxalase -1 across the range of histomorphological spectrum of prostatic lesions can be of use as prognostic allies and indicators of disease progression in adjunct to histological diagnosis.

INTRODUCTION

Prostate is a retroperitoneal fibromuscular gland with a peripheral, central, transitional and periurethral zone. Benign lesions of prostate such as benign prostatic hyperplasia develop commonly in transition zone whereas the precursor lesions of carcinoma prostate; PIN and prostatic carcinomas commonly develop in the peripheral zone of prostate.[1] Prostatic lesions include a wide array of varied histomorphological entities comprising of inflammatory lesions such as prostatitis to benign nodular hyperplasia to prostatic carcinomas and their precursors with incidence known to increase with age.[2]

Benign prostatic hyperplasia is representative of nodular enlargement of gland as a result of hyperplasia of both glandular and stromal components and is known to affect transition zone of prostate most commonly often leading to prostatic

enlargement and urethral obstruction further producing lower urinary tract symptoms.[1,2] Among prostatic carcinomas several histological variants have been observed including Acinar adenocarcinoma, sarcomatoid carcinoma, ductal adenocarcinoma, urothelial carcinoma, squamous and adenosquamous carcinoma, basal cell carcinoma, and neuroendocrine tumours with acinar adenocarcinoma being the commonest variant of all.[3] Both prostatic carcinomas and their precursor lesions; Prostatic Intraepithelial Neoplasia occur most commonly in peripheral zone of prostate.[1]

Prostatic specific antigen is a glycoprotein that has been used as a screening tool for prostatic carcinomas however it is not found to be specific as serum PSA elevations are also observed in acute prostatitis and benign prostatic hyperplasia,[4] while PSMA (prostate specific membrane antigen); an integral membrane protein expresses weakly in normal prostatic tissue and is found to be upregulated in prostate cancer.[5]

Glyoxalase-1, an enzyme of the glyoxalase system works in protective fashion under normal circumstances by detoxification of methylglyoxal (MG); a precursor of advanced glycation end products with potential anti-tumour effect due to its cytotoxic nature however upregulation of GLO-1 has been observed in relation to increased growth and proliferation exhibited in cancer progression along with decreased apoptosis.[6] In our study we aim to study PSMA and Glyoxalase-1 expression in histomorphological spectrum of prostatic lesions ranging from benign lesions to prostatic carcinomas and their precursors in correlation with serum PSA and Gleason Scoring and Gleason Grade group in prostatic carcinomas.

MATERIALS AND METHODS

This cross-sectional study was conducted at Department of Pathology, Subharti Medical College, Meerut between July 2023 and February 2025. A total of 60 prostatic specimens were received comprising of 48 TURP chips, 11 TRUS guided prostatic biopsies and 1 prostatectomy specimen. Informed consent and ethical clearance was obtained for the study. Tissue specimens were subjected to routine histopathology processing, embedding, sectioning & slides were stained with H&E stain and examined under a microscope for histological diagnosis. IHC application with the markers PSMA and Glyoxalase-1 was done using PathSitu rabbit monoclonal antibody and invitrogen rabbit polyclonal antibody respectively. Sections obtained were examined at 4x, 10x and 40x under light microscope for histopathological and immunohistochemical findings.

Statistical analysis: The Statistical Package for Social Sciences (SPSS) software, IBM manufacturer, Chicago, USA, ver 25.0 was used. Presentation of the Categorical variables was done in the form of number and percentage (%). The association of the variables which were qualitative in nature were analysed using Fisher's exact test as atleast one cell had an expected value of less than 5. For statistical significance, p value of less than 0.05 was considered statistically significant.

RESULTS

A total of 60 prostatic specimens comprising of 48 TURP chips, 11 TRUS guided prostatic biopsies and 1 prostatectomy specimen from the patients with the mean age 68.03 years were examined. The most common age group to be affected was 61 -70 years (46.6%) while the youngest patient was 42 years and the oldest patient was 94 years. Majority of the lesions were benign (46 cases, 76.6%) comprising of Benign prostatic hyperplasia with prostatitis (60.0%) and Benign hyperplasia of Prostate (16.6%) followed by 13 cases (21.7%) of prostatic carcinomas including 10 cases of acinar adenocarcinoma, 2 cases of ductal carcinoma and 1case of urothelial carcinoma arising from prostatic urethra with squamous differentiation respectively along with a single case of low grade prostatic intraepithelial neoplasia. Commonest Gleason score was found to be 7 (4+3) followed by score of 9 (4+5) while the commonest Grade group was grade group 3 followed by grade group 5 [Table 1,2].

Table 1: Gleason scoring in prostatic adenocarcinomas

Gleason Score	No. of Cases	Percentage (%)
6 (3 + 3)	1	8.33%
7 (3 + 4)	1	8.33%
7 (4 + 3)	7	58.3%
8 (4+4 / 3+5 / 5+3)	0	–
9 (4+5 / 5+4)	2	16.7%
10 (5 + 5)	1	8.33%
Total (n)	12	100%

Table 2: Gleason Grade group in prostate adenocarcinoma

Gleason Grade Group	No. of Cases	Percentage (%)
1	1	8.33%
2	1	8.33%
3	6	50.0%
4	1	8.33%
5	3	25.0%
Total	12	100%

Compared to serum PSA <10 ng/mL, proportion of patients with serum PSA >10 ng/mL was significantly higher in acinar adenocarcinoma (87.5% vs. 12.5%, p value = 0.0001) while significantly lower proportion was observed in benign prostatic hyperplasia with prostatitis (12.9% vs. 87.09%, p value = 0.019). Comparable proportions were seen in ductal adenocarcinoma (50% in both groups, p value = 0.449), benign prostatic hyperplasia (0%) with PSA>10 ng/mL vs. 100% with PSA <10 ng/mL, p value = 0.096), urothelial carcinoma of prostatic urethra (0% vs. 100%, p value = 1) and BPH with low-grade PIN (100% vs. 0%, p value = 0.255) [Figure 1]

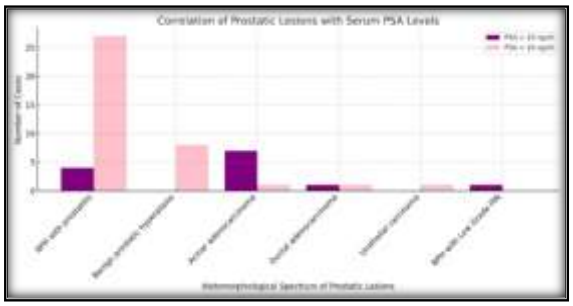

Figure 1: Correlation of Prostatic Lesions with serum PSA levels

Distribution of PSMA expression was comparable across most prostatic lesions: in benign prostatic hyperplasia: inconclusive 0 vs. negative 1 vs. weak cytoplasmic/membranous 3 vs. moderate cytoplasmic 0 vs. strong cytoplasmic 1 vs. cytoplasmic and membranous (strong to moderate) 5 (p value = 0.210); low-grade prostatic intraepithelial neoplasia showed cytoplasmic and membranous (strong to moderate) 1 (p value = 1); in acinar adenocarcinoma: inconclusive 0 vs. negative 0 vs. weak cytoplasmic/membranous 1 vs. moderate cytoplasmic 1 vs. strong cytoplasmic 0 vs. cytoplasmic and membranous (strong to moderate) 8 (p value = 0.186); urothelial carcinoma of prostatic urethra showed weak cytoplasmic/membranous 1 with no other expression (p value = 0.600).

In benign prostatic hyperplasia with prostatitis, PSMA expression showed significant distribution, with higher values for weak cytoplasmic/membranous (13) and cytoplasmic and membranous (strong to moderate) (10) compared to inconclusive 0, negative 4, moderate cytoplasmic 9 and strong cytoplasmic 0 (p value = 0.040) and Ductal adenocarcinoma also showed significant distribution, with PSMA expression observed only in inconclusive (1) and moderate cytoplasmic (1), with no other expression (p value = 0.024) [Figure 2]

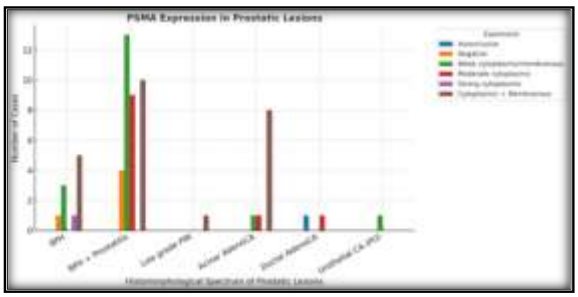

Figure 2: Correlation of PSMA expression with histomorphological spectrum of prostatic lesions.

Distribution of Glyoxalase-1 expression was comparable across most prostatic lesions: in benign prostatic hyperplasia: inconclusive 0 vs. negative 4 vs. weak cytoplasmic 4 vs. moderate cytoplasmic 1 vs. cytoplasmic and nuclear (weak to moderate) 1 (p value = 0.411); in benign prostatic hyperplasia with prostatitis: inconclusive 2 vs. negative 7 vs. weak cytoplasmic 17 vs. moderate cytoplasmic 8 vs. cytoplasmic and nuclear (weak to moderate) 2 (p value = 0.645); low-grade prostatic intraepithelial neoplasia showed moderate cytoplasmic expression 1 (p value = 0.583); in acinar adenocarcinoma: inconclusive 1 vs. negative 1 vs. weak cytoplasmic 4 vs. moderate cytoplasmic 3 vs. cytoplasmic and nuclear (weak to moderate) 1 (p value = 0.879) and urothelial carcinoma of prostatic urethra showed cytoplasmic and nuclear (weak to moderate) expression 1 (p value = 0.167), while ductal adenocarcinoma showed significant finding with inconclusive 2 and no other expression (p value = 0.011) [Figure 3].

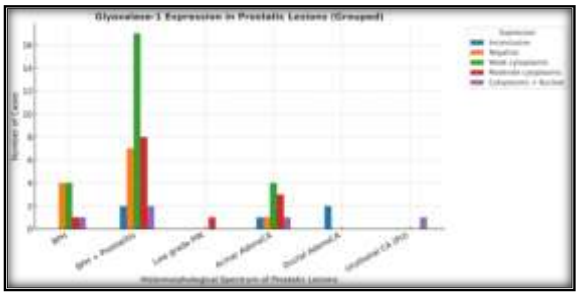

Figure 3: Correlation of Glyoxalase -1 expression with histomorphological spectrum of prostatic lesions.

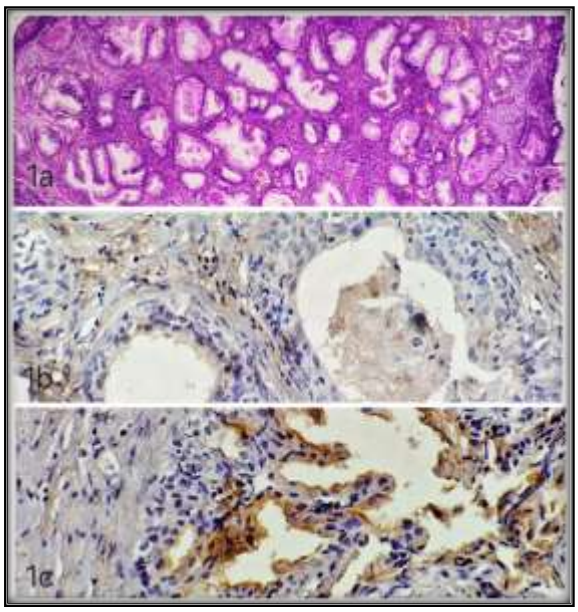

Image 1a: Benign prostatic hyperplasia (BPH), 1b: Negative Glyoxalase-1 expression in BPH, 1c: Weak membranous PSMA expression in BPH

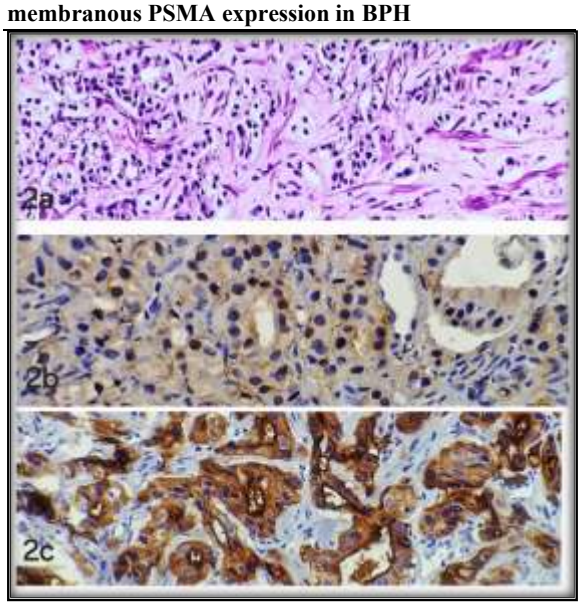

Image 2a: Prostatic adenocarcinoma Gleason score 7(4+3), Gleason Grade group 3 (H&E, 40X), 2b: Weak cytoplasmic Glyoxalase-1 expression (40X), 2c: Strong membranous and cytoplasmic PSMA expression

Strong membranous and cytoplasmic PSMA expression was seen in association with 4/7 cases of prostatic adenocarcinoma with Gleason score 7(4+3) as opposed to moderate cytoplasmic expression in adenocarcinoma with Gleason score 7(3+4), however Glyoxalase -1 expression was found to be weak cytoplasmic in 3/7 prostatic adenocarcinoma cases with Gleason scoring 7 (4+3). Similarly strong membranous and cytoplasmic PSMA expression was observed in 3/6 cases with most frequent Grade Group 3 while weak cytoplasmic Glyoxalase -1 expression was seen in majority of the cases assigned grade group 3 [Table 3,4].

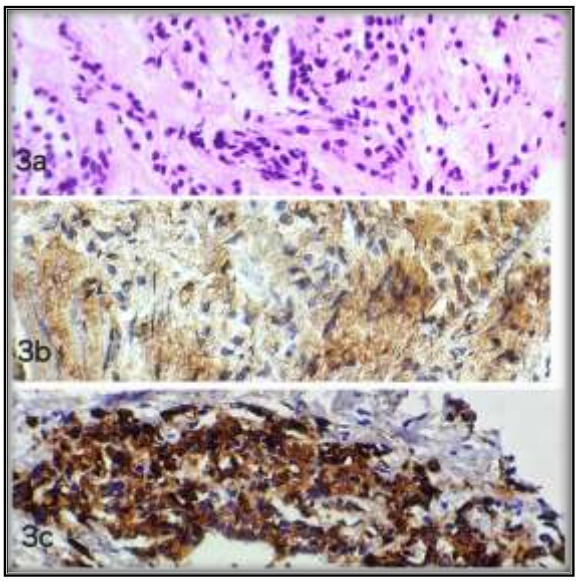

Image 3a: Prostatic adenocarcinoma, Gleason score 9 (4+5), Gleason grade group 5 (H & E, 40X), 3b: Moderate cytoplasmic Glyoxalase-1 expression (40X), 3c: Strong membranous and cytoplasmic PSMA expression.

Table 3: PSMA and Glyoxlase-1 expression across various Gleason scores

Gleason score	PSMA Expression	Glyoxalase – 1 Expression
6 (3+3) (n = 01)		Moderate cytoplasmic positivity
7 (3+4) (n = 01)	Moderate cytoplasmic positivity	inconclusive
7 (4+3) (n = 07)	Strong membranous and cytoplasmic positivity = 04 Moderate cytoplasmic positivity = 01 Weak cytoplasmic positivity = 01 Inconclusive = 01	Moderate cytoplasmic positivity = 01 Moderate cytoplasmic and nuclear positivity = 01 Weak cytoplasmic positivity = 03 Inconclusive = 02
8 (4+4/3+5/5+3) (n = 00)		
9 (4+5/5+4) (n = 02)	Strong membranous and cytoplasmic positivity	Moderate cytoplasmic positivity = 01 Negative = 01
10 (5+5) (n = 01)	Strong membranous and cytoplasmic positivity	Weak cytoplasmic positivity

Table 4: PSMA and Glyoxalase -1 expression across various Gleason Grade Groups

Gleason Grade Group	PSMA Expression	Glyoxalase-1 Expression
1 (n = 1)	Strong membranous and cytoplasmic positivity	Moderate cytoplasmic positivity
2 (n = 1)	Moderate cytoplasmic positivity	Inconclusive
3 (n = 6)	Strong membranous & cytoplasmic positivity = 3 Moderate cytoplasmic positivity = 1 Weak cytoplasmic positivity = 1 Inconclusive = 1	Moderate cytoplasmic & nuclear positivity = 1 Weak cytoplasmic positivity = 3 Inconclusive = 2
4 (n = 1)	Strong membranous and cytoplasmic positivity	Moderate cytoplasmic positivity
5 (n = 3)	Strong membranous and cytoplasmic positivity	Strong membranous and cytoplasmic positivity = 1 Weak cytoplasmic positivity = 1 Negative = 1

Thus it was deduced that PSMA expression was in direct proportionality with increase in serum PSA levels (> 10ng/mL) along with increasing Gleason Score and Grade group while there was considerable variability in Glyoxalase -1 expression ranging from prostatic intraepithelial neoplasia to prostatic carcinomas and in accordance with serum PSA levels, Gleason Score and Gleason Grade group.

DISCUSSION

Prostatic lesions encompass a spectrum from inflammatory conditions to carcinoma. Our study evaluated immunohistochemical expression of PSMA and Glyoxalase-1 (GLO-1) across this spectrum. Sharma A et al,[7] reported benign nodular hyperplasia as the most common diagnosis (223/245 cases), followed by PIN (14 cases) and adenocarcinoma (8 cases). Similarly, Satyasri K et al,[8] observed BPH in 279/321 cases (with associated prostatitis in 117 cases), prostatic carcinoma in 27 cases, and PIN in 15 cases. In our study, out of 60 specimens, BPH was seen in 46 cases, prostatic carcinoma in 13, and PIN in 1 with mean age of presentation being 68.03 years which was comparable to age of presentation observed by Ngugi PM et al,[9] Ojewola RW et al,[10] Hameed S et al,[11] and Mishra SKC et al.[12]

Serum PSA was available in 51/60 cases, with mean serum PSA being 13.31 ng/ml. Most benign cases had PSA <10 ng/ml (except in 4 cases). Of 11 malignant cases, 8 showed serum PSA levels >10 ng/ml (maximum level being >100). The single case of PIN also had serum PSA levels >10 ng/ml. Similar findings were reported by Naskar S et al,[13] and Garalla et al.[14]

Of 13 malignant cases, 12 were prostatic adenocarcinoma and 1 was urothelial carcinoma arising from prostatic urethra with squamous differentiation. Modified Gleason scoring in prostatic adenocarcinomas revealed Gleason 7 (4+3) in 58.3% (7/12 cases), followed by Gleason Score 9 in 16.7%, (2/12 cases). Grade Group 3 was most common (6/12 cases), followed by Group 5 (3/12 cases) and 1 case of Grade Group 1,2 and 4 each. Comparable results were reported by Rani et al,[15] in 35% (28/80 cases) in Grade Group 3 along with Gleason score 7 [4+3].

PSMA expression in benign lesions was largely absent to weak, with the majority demonstrating either weak cytoplasmic/membranous or combined cytoplasmic and membranous staining. This finding is consistent with previous observations by Bravaccini et al,[16] who reported minimal PSMA expression in benign prostatic tissue, thereby reinforcing its potential role as a marker of neoplastic transformation. Notably, the single case of low-grade PIN in our study exhibited moderate membranous and cytoplasmic PSMA expression, supporting prior evidence that PSMA upregulation may begin at the pre-neoplastic stage. Among malignant lesions, acinar adenocarcinoma predominantly demonstrated combined membranous and cytoplasmic PSMA expression, with higher intensity correlating with tumor aggressiveness. The presence of strong PSMA positivity in high-grade lesions, including those with Gleason scores of 9 and Grade Group 5, is in concordance with Bravaccini et al,[16] who reported stronger PSMA staining in higher Gleason patterns (4 and 5) compared to lower-grade tumors.

GLO-1 expression showed a different distribution pattern, with benign lesions primarily demonstrating weak cytoplasmic staining and a substantial proportion being negative. Interestingly, moderate GLO-1 expression was observed in low-grade PIN, and malignant lesions exhibited variable positivity. Acinar adenocarcinomas showed predominantly weak to moderate cytoplasmic expression, with occasional nuclear localization, whereas ductal adenocarcinomas were inconclusive. The single urothelial carcinoma case demonstrated moderate cytoplasmic and nuclear staining. These findings align with those of Rounds et al,[17] who documented GLO-1 expression in both PIN and prostate carcinoma, with maximal expression observed in intermediate-grade tumors such as Gleason score 7 (4+3). The variable expression in high-grade tumors in our cohort suggests that while GLO-1 may play a role in early tumorigenesis, its expression does not necessarily correlate linearly with increasing tumor grade.

Correlation of immunohistochemical findings with Gleason score and grade group further highlighted the prognostic relevance of PSMA. In our study, strong membranous and cytoplasmic PSMA expression was predominantly associated with higher Gleason scores and grade groups, reinforcing its potential utility as a biomarker for aggressive disease. Conversely, GLO-1 expression showed no consistent trend with tumor grade, being weak in many

intermediate-grade tumors and variable in high-grade lesions. This supports the hypothesis that while PSMA may serve as a reliable marker of tumor aggressiveness, GLO-1 could be more indicative of early neoplastic change rather than progression.

Overall, our findings support the growing body of evidence that PSMA is a highly specific marker for prostate cancer progression and aggressiveness, while GLO-1 may have diagnostic utility in identifying early neoplastic lesions such as PIN. These observations, consistent with the reports of Bravaccini et al,[16] and Rounds et al,[17] emphasize the complementary roles of these biomarkers in the histopathological evaluation of prostatic lesions and suggest their potential integration into routine diagnostic and prognostic assessment.

CONCLUSION

PSMA expression increases with the progression from benign to malignant prostatic lesions and correlates strongly with higher Gleason scores and grade groups, reinforcing its role as a potential marker for tumor aggressiveness. GLO-1, while variably expressed in malignant lesions, shows significant positivity in preneoplastic changes such as PIN, indicating its role in early tumorigenesis rather than disease progression. Together, PSMA and GLO-1 may serve as valuable complementary biomarkers in the diagnosis, risk stratification, and potential therapeutic targeting of prostatic lesions. Further large-scale studies are warranted to validate these findings and explore their integration into clinical practice.

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