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Original Research Article

Immunohistochemistry analysis of estrogen receptor and androgen receptor in benign prostatic hyperplasia and prostate carcinoma

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ABSTRACT

Background: Benign prostatic hyperplasia (BPH) and prostate adenocarcinoma are the most common disorders affecting elderly men. The diseases are androgen-dependent and are treated by obstructing androgen receptor (AR) or their action. AR has direct relationship between the development of prostatic hyperplasia and prostatic carcinoma. Estrogens directly target prostate tissue by definite estrogen receptors (ER). The human prostate is primed with a dual system of ERs (ER-α and ER-β). Use of estrogens as hormonal agents is for suppressing prostatic tissue growth. Study aims to identify, assess and establish the significance of ER and AR as a marker in distinguishing BPH and prostatic carcinoma.

Materials and Methods: A cross sectional study was conducted at a tertiary care hospital over a period of two year on 65 specimens which included 30 malignant cases and 35 benign cases. Specimens received were tru-cut biopsy, transurethral resection of prostate chips (TURP) and prostatectomy specimen for malignant cases and TURP for benign lesions. Paraffin embedded blocks was stained with H&E and IHC with ER and AR performed on representative sections and graded .the results were assessed for association with various parameters in both benign and malignant cases.

Results: Out of 30 cases of adenocarcinoma of prostate majority of specimens were needle biopsies (54%). The age of presentation was 55-84 years with mean age being 68.93 years. In cases of BPH the presenting age was 35-84 years and mean age was 63 years. Histopathological parameters assessed for malignant lesions, showed negative correlation of grade group with PNI and positive correlation with neutrophils. Higher grade showed adverse histological parameters. %PSA was <25% in malignancy. ER positivity was noted in 80% malignant and 100% benign cases with weaker positivity noted maximum in benign cases. AR positivity was noted in all cases both benign and malignant, with benign cases showing higher intensity.

Conclusions: Hormones play a multifactorial role in the pathogenesis of BPH and prostatic carcinoma; most commonly androgen and estrogen, and can be used towards finding a better therapy.

1. Introduction

Benign prostatic hyperplasia (BPH) and prostate adenocarcinoma (PCa) are the most common diseases affecting the prostate in elderly men. PCa is the 16th most commonly diagnosed cancer in men and 16th leading cause of cancer-related death.[1] Numerous studies have focused on the association between androgens to PCa and BPH risk, have suggested these are androgen-dependent, with treatment by obstructing androgen receptor or their action. Androgens drive prostatic growth and thence development of prostatic hyperplasia and carcinoma, mediated by specific androgen receptors (AR).[2]

Early work on the hormonal basis of prostate cancer focused on the role of androgens, but more recently

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estrogens have also been postulated as potential agents in the development and progression of prostate cancer, with substantial work towards estrogen signaling as playing a momentous role in normal and abnormal growth of the prostate gland. Estrogens directly target prostate tissue by definite estrogen receptors (ER). The human prostate is primed with a dual system: ER-α and ER-β.[3] Estrogen receptors are only present in minute proportion of prostatic tissues. The mainstream of PCa and BPH specimens exhibited nuclear immunoreactivity for ER-β in both tumor and stromal cells.[3] The uses of estrogens as hormonal agents are to suppress prostatic tissue growth. It is therefore less likely to achieve therapeutic goal, unless the estrogen receptor status is known.

The development of prostate cancer usually occurs at an age when the level of serum testosterone is decreasing. In contrast, the levels of estradiol do not diminish and as a substitute this remains unchanged or increases with age. Earlier results suggest that the momentous reduction in ratio of testosterone to estradiol is related to PCa development. PCa is an androgen-sensitive disease, which can be pharmacologically prohibited by androgen blockade.[4] This study aims to assess the implication of ER and AR as biological markers in assessing their degree of positivity in Benign prostatic hyperplasia (BPH) and Prostatic carcinoma (PCa), and to determine their association with PSA levels and histopathological morphology.

2. Materials and Methods

A Cross sectional study which was conducted in the Department of Pathology at a Yenepoya Medical College over a two year period on 65 prostatic specimens, after obtaining ethical clearance from the university with approval reference number 2017/200. These included histopathologically diagnosed cases of prostatic carcinomas (30 cases) on prostate biopsy/ TURP chips/ prostatectomy specimens and benign prostatic hyperplasia (35 cases) diagnosed on TURP chips were incorporated. ER and AR was performed on formalin fixed, paraffin-embedded tissue sections of the most appropriate tumor block selected.Primary Kit: ERα clone: EP1, Flex monoclonal mouse antihuman ER, DAKO. AR: clone AR441 monoclonal mouse anti-human AR protein, DAKO Secondary Kit: DAKO Real Envision were utilized.

IHC Reporting for Androgen receptor (AR): All AR IHC stained slides were evaluated for nuclear staining and were scored for AR expression. Scoring of intensity and percentage positivity was carried out separately.[4,5] Staining intensity was scored from 0-3 as shown in Figures 1, 2 and 3. Where staining intensity is graded as 0= not detectable, 1= translucent, 2= opaque, 3= solid.[4,5] The percentage of cells staining positively at each level of staining intensity was calculated. We used total staining score, defined as the sum of the percentage of any cells that are staining positively. We also evaluated other IHC scoring functions, including the H score, to determine whether they may perform better in classifying responders.[4–6] The H score is a widely used, semi quantitative measure that considers both the cell staining intensity and the percentage of cells stained positively.[5] The score is obtained by the equation = 3 x score 3 (percentage of solid staining nuclei) + 2 x score at 2 (percentage of opaque-staining nuclei) + score 1(percentage of translucent-staining nuclei), yielding results in a range of 0 to 300, which are then normalized to a scale of 0% to 100%.,[5] as described in Figure 4.

IHC Reporting for Estrogen receptor (ER): The staining results obtained by IHC analysis were classified into six categories ranging from 0 to 5.,[3,7] as shown inFigures 5, 6, 7 and 8. Where Category 0: no staining detectable; Category 1: less than 5% positive tumor cells; Category 2: 5-25% positive tumor cells; Category 3: 25-50% positive tumor cells; Category 4: 50-75% positive tumor cells; Category 5: More than 75% positive tumor cells.

The data obtained was entered into excel sheet and analyzed using the statistical package for IBM Corp. Released 2017. IBM SPSS Statistics for Windows, Version 25.0. Armonk, NY. Frequencies and percentage of all variables were computed. The data thus analyzed, were correlated for relevant findings. Chi- square paired t test was performed and a p value of <0.05 was considered statistically significant.

3. Results

The mean age of distribution for malignant cases was 68 years with maximum and minimum age of presentation as 84 and 55 years with standard deviation of 6.223. Whereas in BPH the mean age of distribution was 63 years, with maximum age of presentation as 85 years, minimum 39 years, SD of 8.118. It was noted that the maximum number of patients in categories belonged to the 7th decade. The predominant type of specimen received for malignant cases were true cut biopsy (54%) followed by TURP and prostatectomy specimens which were 23% each. For benign lesions only TURP chips were selected in our study. On subcategorizing the benign lesions, it was found that BPH and BPH with chronic inflammation was seen in 37% cases followed by BPH with acute on chronic inflammation (17%) and BPH with acute inflammation (6%) cases.

3.1. PSA levels analysis

Out of 30 carcinoma cases, PSA levels were recorded for 25 carcinoma cases. No PSA records were available for five cases. The minimum and maximum tPSA level for malignant cases was 5.35 ng/mL and 1564 ng/mL with a mean of 180.5 ng/mL ± 417.9. The minimum and maximum fPSA levels were 0.673 ng/mL and 180.4ng/mL with a mean

of 31.3ng/mL ±49.3. The %PSA ranged from 2% to 42% with a mean of 12% ± 10.8%. Maximum number of cases (40%) showed a high tPSA levels >70 ng/mL. 36% of cases showed tPSA level >15 ng/mL. However, only 24% of case showed lower tPSA level of 5 to15 ng/mL. fPSA levels were assessed in 25 carcinoma cases. Maximum number of cases (64%) showed fPSA levels of >3ng/mL. Taking a level of 25% as a cutoff value in carcinoma cases for %PSA, it was noted that maximum number of cases with <25% value was seen in 80% of cases and >25% percentage PSA level was noted in only 20% of cases.

When age was correlated with various PSA levels such as tPSA, fPSA and %PSA in malignant lesions, it showed a strong positive correlation with %PSA levels (p value: 0.029) and a negative correlation with fPSA levels (p value: -.047). No correlation was seen between tPSA and age in malignant lesions. Maximum number of cases ranged between age limit 60-70 years with tPSA level 61-100 ng/mL. The %PSA of <25% in malignant cases were noted in the 6th and 7th decade of life, and was observed in 67% cases. There was no correlation between PSA level and grade group, Gleason's score and stage of the carcinoma.

Out of 35 benign cases, PSA levels were recorded for 30 benign lesions. No PSA records were available for five cases. The minimum and maximum tPSA level for benign lesions was 0.233 ng/mL and 31.3 ng/mL with a mean of 5.57 ng/mL ±7.89. Whereas the mean fPSA levels in benign cases is 1.24ng/mL ± 1.53. The %PSA mean was of 19.1%±10.09%. There was a strong positive correlation between tPSA levels and age in benign cases (p value: 0.041).

3.2. Histopathological data analysis

In the present study, 50% carcinoma cases showed Gleason's score of 9 followed by Gleason's score 7(23%) and 8(17%). The maximum number of prostatic carcinoma cases showed grade group 5(57%) followed by grade group 4(20%), grade group 2(13%) and grade group 3(10%). Various histopathological parameters studied in carcinoma cases are necrosis, PNI, lymphatic invasion, PIN, inflammatory infiltrate of lymphocytes and neutrophils. When correlated with Grade group and Gleason's score there was a positive correlation between grade group and neutrophils (p value: 0.0053) and negative correlation between grade group and perineural invasion (p value:-0.042). However no correlation was seen between Gleason's score and histological parameters. Percentage presence and absence of the respective parameters are presented in Table 1.

However benign lesions showed more of stromal component in comparison to glandular component. Stroma >50% was seen in 46% cases and glands >50% were seen only in 37% cases. Moderate (=50%) glands and stroma were seen in 17% of cases. Adenosis was noted in 57% of cases. Inflammatory infiltrate such as lymphocytes was seen in of 89% of cases, whereas 60% of cases showed neutrophils. Gland destruction by foam cells was seen in 69% of cases. Granuloma was seen in one case with PSA level of 3ng/mL. There was no clear cell change noted in the present study.

3.3. Estrogen receptor and its assessment in carcinoma cases

IHC ER was assessed in 30 malignant cases, out of which 80% cases showed positive ERα as shown in Table 2. Carcinoma cases showed 71% of stromal ERα positivity, 25% cases ERα positivity in combined glandular and stromal tissue and only glandular staining was seen in 4% of cases. Grade group 5 showed maximum ERα positivity which was seen in 45% cases, which included only stromal ERα positivity in 63% cases and 36% cases showed combined glandular and stromal ERα positivity. Purely stromal and combined glandular and stromal ER positivity increased with increase in grade group. There is a strong positive correlation between grade group and % ER positivity (p=0.015).

On correlating with ER positive malignant lesions with various clinical parameters like age and PSA levels and there was positive correlation with age however no correlation was noted with PSA levels. Similarly when correlated with prognostic parameters, it was seen that tumor percentage had strong positive correlation with % ERα positivity (p value: 0.033) and showed a negative correlation with presence of lymphatic emboli (p value: -0.035). There were no correlations between other parameters such as PNI, stage, necrosis, neutrophils and PIN.

3.4. Androgen receptor (AR) positivity and its assessment in carcinoma cases

AR was assessed in 25 carcinoma cases out of 30 cases. Five cases were not assessed due tissue exhaustion. 60-100% glandular and stromal AR positivity was seen in 32% of cases. 30-60% glandular and stromal AR positivity was seen in 28% of cases. 10 to 30% and <10% glandular and stromal AR positivity were seen in 20% of cases as shown in Table 3. Correlation of various parameters with AR expression was assessed. However, none of the parameters showed statistical significance with p value <0.05

3.5. Estrogen receptor and Androgen receptor positivity and its assessment in benign cases

ER positivity was assessed in 35 benign lesions as shown in Table 4. There was an inverse relation noted between the two. 49% of benign cases showed gland with stromal staining, which was maximum. Purely stromal positive

staining was seen in 34% of cases and 17% of cases showed purely glandular staining.

Percentage AR positivity analysis was done on 35 benign cases. More than 60% staining was seen in 77% of cases whereas 23% of cases showed <60% staining. Paired t test was performed to assess the correlation between %ER and %AR positivity in benign cases. Strong positive correlation was identified with p value of 0.039.

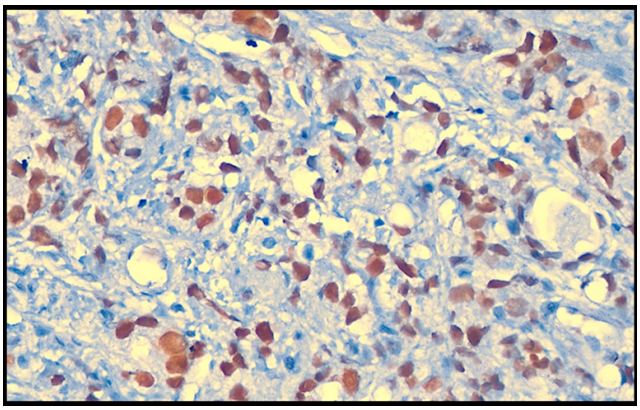

Fig. 1: Photomicrography of adenocarcinoma prostate showing strong intensity (score 3- solid) AR positive stained glandular cells. (40X)

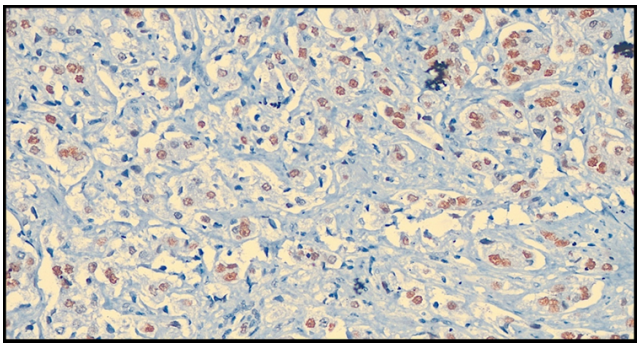

Fig. 2: *Photomicrography of prostatic adenocarcinoma showing score 2 (opaque) and score 3 (solid) nuclear AR positivity. (40x)*

Table 1: Percentage presence and absence of histological parameters

Parameters	Present	Absent
Necrosis	23%	77%
PNI	47%	53%
Lymphatic invasion	30%	70%
PIN	13%	87%
Lymphocytes	100%	-
Neutrophils	57%	43%

PNI: Perineural invasion, PIN: Prostatic intraepithelial neoplasia

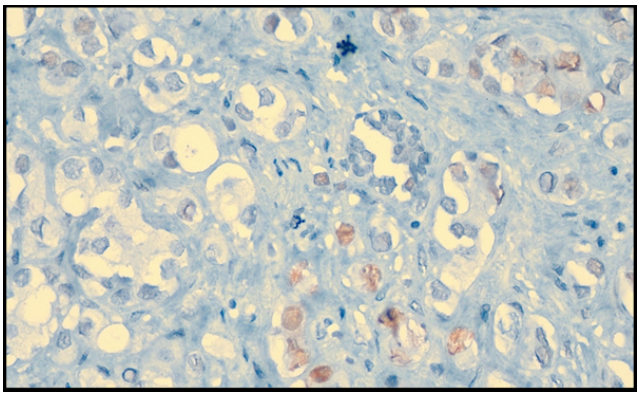

Fig. 3: *Photomicrography of prostatic adenocarcinoma showing score 1 (transparent) nuclear AR positivity (40x).*

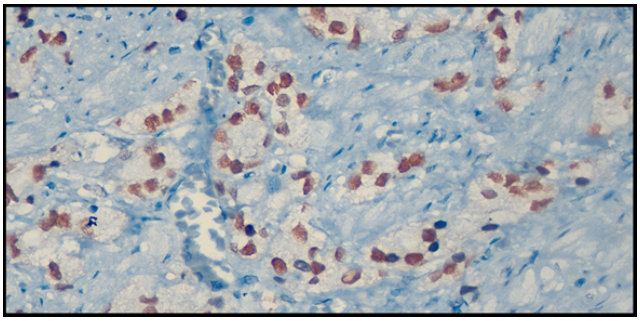

Fig. 4: *Photomicrography showing benign lesion with 87% AR positivity (40x)*

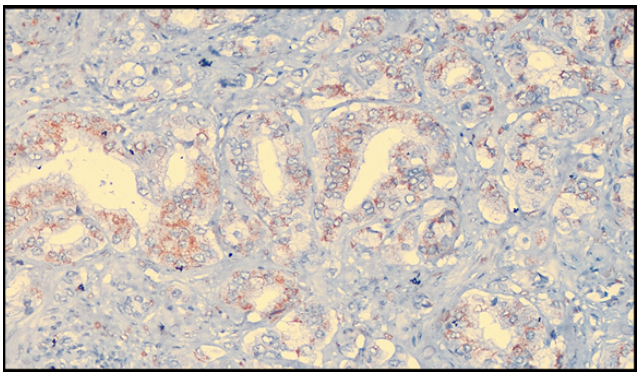

Fig. 5: *Photomicrography showing Grade 1 ER glandular positivity in Prostatic carcinoma (40x)*

Table 2: Estrogen receptor positivity in malignant cases

Category	Percentage malignant cases positivity
Category 0(no stain)	20%
Category 1(<5%)	30%
Category 2(5-25%)	30%
Category 3(25-50%)	20%
Category 4 and 5 (>50%)	0%

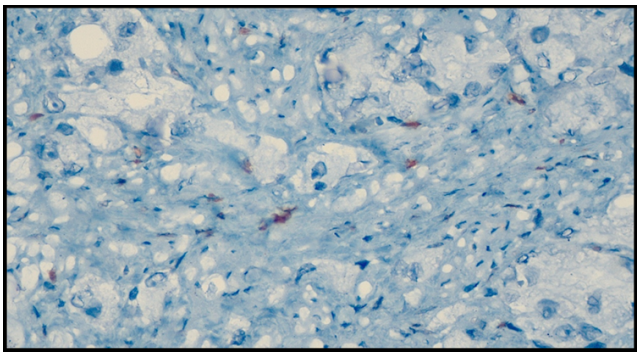

Fig. 6: *Photomicrography showing Grade 1 ER stromal positivity in Prostatic carcinoma (40x)*

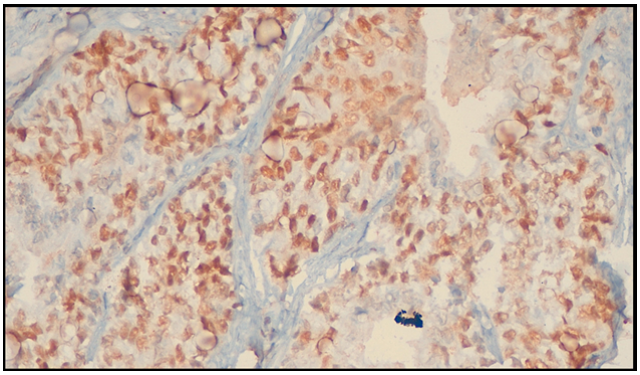

Fig. 7: *Photomicrography showing Grade 3 ER glandular positivity in benign lesion (40x)*

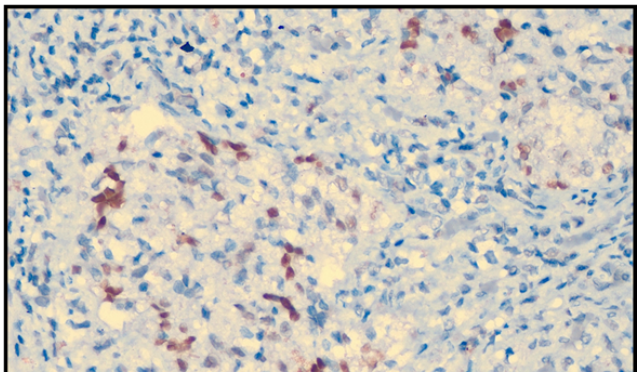

Fig. 8: *Photomicrography showing Grade 1 ER stromal positivity in benign lesion (40x)*

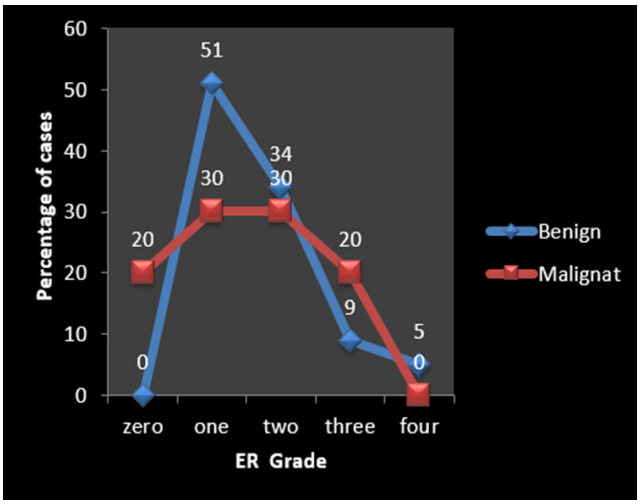

Fig. 9: Demonstration of ER expression in BPH and Carcinoma cases

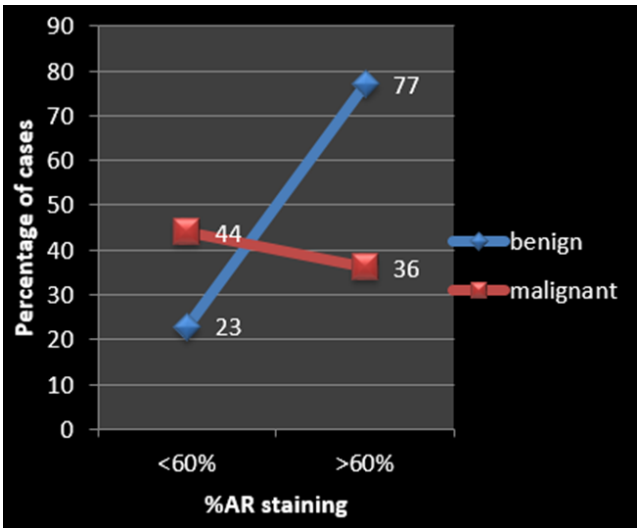

Fig. 10: Demonstration of AR expression in BPH and Carcinoma cases

Table 3: Androgen receptor positivity in malignant cases

Percentage staining of Androgen receptor	Percentage malignant cases positivity
<10%	20%
10-30%	20%
30-60%	28%
60-100%	32%

Table 4: Estrogenreceptor positivity in benign cases

%ER positivity	No. of cases
<5%	18(52%)
5-25%	12(35%)
25-50%	3(8%)
50-75%	2(5%)

4. Discussion

According to latest GLOBOCAN 2018, prostate carcinoma is 16th most common carcinoma to cause death among both the sexes accounting to 2.2% new cases.[6] Benign prostatic hyperplasia (BPH) and prostate adenocarcinoma (PCa) are the most common diseases affecting the prostate in elderly men.[7]

For the development, growth and maintenance of the prostate androgens are essential. The advancement and progression of both BPH and PCa is by the action of androgens on prostatic tissue. The evolution of PC is probably to be the effect of an abnormal AR status. Androgen is an important hormone in pathogenesis of prostate cancer. They attach to the androgen receptors and provoke the expression of pro-survival and pro-development.[7] An indirect anti-androgen factor was noted by estrogen hormone. Estrogens have two types of receptor (ERα and ERβ), which are capable of appreciably influence PC growth and development. Both receptors are articulated in the adult prostate, ERα expression appears to be restricted to the stromal compartment and ERβ is present in both the epithelial cells and to a lesser extent in the stromal compartment.[8]

In our study the age of presentation for malignant lesions ranged from 55 to 84 years with a mean of 68 years was similar to study done by Aslam HM et al and Hameed S et al.[7,9] In benign lesions age ranged from 39 to 85 years with a mean age of 63 years. Most of the patients were in 7th decade of life for both benign and malignant lesion. These findings were supported by study done by Hameed S et al and Mansoor I et al.[9,10] Aslam HM et al stated that frequency of hyperplasia increases with age from fifth decade to seventh decade, this reflects that hyperplasia may be a normal aging process.[7] According to the study by Naskar S et al mean age of presentation for prostatic lesions were 68.66 years[11] which was analogous to our study. There were no significant age differences detected between the benign and malignant cases as stated by Men S et al, our study was also in concordance with the study conducted by Men S et al where the mean age distribution for prostatic lesions is 64.67 years.[12] Serum tPSA levels in 30 carcinoma cases ranged from 5.35 to 1564ng/ml with mean of 180.48ng/ml whereas in BPH (n=35) tPSA ranged from 0.23 to 31.30 ng/ml with mean of 5.5ng/ml including cases of inflammation. Chopra GS et at in their study stated that in 48 cases of carcinoma prostate PSA level ranged from 8.4 to 260 ng/mL with mean 74.75 ± 59.39 ng/mL and in BPH PSA levels ranged from 0.2 to 14.5 ng/mL (mean 4.66 ± 3.85).[13]

Up till now, the Gleason score and grade groups are strong predictors for disease progression, and the most important parameters in therapeutic decision-making.[14] In our study, 50% of cases showed Gleason's score 9 followed by Gleason's score 7 (23%) and score 8 (17%). KirakoyaB et al and Hoogland AM et al in their study recorded that 72% (1143/1577) cases showed Gleason score 7 on biopsy and radical prostatectomy followed by Gleason score 8. Gleason score 9-10 on needle-biopsies were 58% (69/119) had a similar Gleason score on radical prostatectomy.[15,16] In our study, Gleason's grade group 5 was seen in maximum number of cases (57%). Bhatta S et al stated most common grade group in their study was grade group 4 and the most predominant Gleason score was 9, which indicated poor prognosis.[17] Gleason score of 9 was seen in 3 out of 8 cases (37.5%). A study by Deshmukh BD et al found Gleason score 9 in 33.33% cases.[18] Bhat S et al observed 56.16% cases of adenocarcinoma with Gleason score of 8-9 which was similar to our study.[19] In a study by Kasliwal N et al Gleason score 8 was the most common score seen in 47.4% cases.[20] Various histopathological parameters were included in our study for malignant lesions; necrosis was seen in 23% cases, PNI was present in 47% cases which were similar to study by Lubig S.et.al where PNI is found in 53.5% of cases.[21] Bhatta S et al in their study found PNI in 37.5% cases.[17] In our study, lymphovascular invasion (LVI) was noted in 30% case and high Gleason's score 9 (20%). May M et al in their study stated that LVI was seen in 10.2% cases with high PSA, 79% of cases had a Gleason score of > or = 7.[22]

Immunohistochemical Estrogen Receptor α (ER α) analysis was done on 30 malignant cases of prostate and 35 benign cases. The staining results obtained by IHC analysis were classified into six categories ranging from 0 to 5. 80% of cases were positive for ER staining in malignant cases in our study. Grade 0 and grade 3 positivity was seen in 20% of malignant cases. In 30% of cases Grade 1 and Grade 2 staining was observed. The staining was seen only till grade 3 which comprised of 25-50% staining of ER. Higher grades 4 and 5 were not observed. Al Magharabi et al in their study stated that out of 65 carcinoma cases only 4.6% cases revealed nuclear immunoreactivity whereas 95.4% cases were negative for ER expression.[3] In our study Grade group 5 showed maximum ERα positivity which was seen in 45% cases, which included only stromal ERα positivity in 63% cases and 36% cases showed combined glandular and stromal ERα positivity. Purely stromal and combined glandular and stromal ER positivity increased with increase in grade group. There is a strong positive correlation between grade group and % ER positivity (p=0.015). There was a weak correlation between glandular and stromal staining with grade group (p- 0.089) in malignant lesions. Bonkhoff H et al in their study stated that in primary tumors, the extent of detectable ER in epithelial compartments correlated significantly with the primary Gleason grade.[8] Low to intermediate grade adenocarcinoma expressed the ER protein in a minority of cases.[8] On the other hand, high-grade (Gleason grades 4 and 5) tumors revealed least focal ER positivity in 43% of cases. Purely epithelial component

staining was seen in 4% of cases. These were not seen in higher grade group, however gland with stromal staining was seen in 25% of cases and purely stromal staining which was seen in 71% of cases were noted in higher grade group (grade group 4 and 5). There was a negative correlation seen in our study between percentage ER staining and Gleason's score (P value: -.018) and positive correlation with tumor percentage (p value: 0.033). Magharabi AL et al in their study stated that two of ER positive cases were of Gleason's score 7 and Gleason's score 8, 6.2% of cases exhibited stromal cell nuclear immunoreactivity and 10.8 % of cases demonstrated weak non- specific cytoplasmic staining and no nuclear staining was seen.[3]

Immunohistochemically when assessed for ER staining for benign lesions in our study showing Grade 1(<5%) staining was seen in 51% of cases where as Grade 4 (50 to 75%) staining was seen only in 6% of cases and no cases was seen with Grade 5 (>75%) staining. Glandular and stromal staining was seen in 49% of cases where as purely stromal staining was observed in 34% of cases and only epithelial staining was seen in 17% of cases. However in a study by Maghrabi Al stated in their study that all BPH specimens were negative for ERα immunoexpression in epithelial cells, although 4 BPH (11.4%) showed nuclear stromal reactivity for ERα.[3] Hetzl et al. reported intense immunoreactivity for ERα and weak immunoreactivity for ERβ in the epithelium of BPH.[23] Nicholson et al. demonstrated that BPH and normal prostate had a similar percentage of ERα positive cells overall, resulting from an increased expression of ERα in epithelial cells but decreased expression in stromal cells in BPH.[24] There was no correlation noted between BPH with inflammation and % ER staining (p value: 0.266).

In our study ER expression was seen in all cases of BPH and 80% carcinoma cases as described in figure 9. However Naskar S et al showed negative ER expression in prostate tissues and was negative in all cases of BPH and prostate carcinoma.[11] These IHC results were identical to that of other studies such as Wernert et al. Where they found the ER was demonstrated in nuclei of periglandular fibrocytes, smooth muscle cells and hyperplatic basal cells, but glandular secretory epithelium were negative in prostatic carcinoma cases.[25]

Immunohistochemically Androgen Receptor (AR) analysis was done on 30 malignant cases of prostate and 35 benign cases. More than 60% staining was seen in 77% of cases whereas 23% of cases showed <60% staining in BPH. More than 60% staining was seen in 36% of cases whereas 44% of cases showed <60% staining in prostatic carcinoma as described in figure 10. Khalid BA et al in his study stated that of 9 prostatic carcinoma cases 6 (66.7%) were positive for androgen receptors.[26] Husain et al in his study stated that intensity of AR staining in cases of adenocarcinoma was moderate in four cases (40%) and strong in six cases (60%) [27]. Kobayashi PE et al stated in their study that four cases of PC were negative for AR and two tumors showed lower expression (less than 25% of positive cells).[28] There was no correlation between AR and other parameters such as Grade group, Gleason's score, tumor percentage, necrosis, TNM staging. Husain et al also stated in their study that there was no significant statistical association between the AR expression and tumor, necrosis, metastasis and TNM stage.[27]

AR immunoreactivity when assessed in benign lesions showed >60% staining in 77% of cases where as <60% staining in 23% of cases. Khalid BA et al in their study stated that out of 97 BPH tissue, 87 were positive (89.7%). And intensity of nuclear staining was seen more in benign in compared to malignant cases was similar to our study.[26] However in one study by Husain I et al stated that the staining was more intense in cases of adenocarcinoma and PIN as compared to BPH.[27] Filipovski v et al stated the correlation between AR expression in the epithelial and stromal cells of BPH is positive but statistically not significant.[4] Nicholson et al. resorted to the method of multiplexed IHC, which revealed an increased percentage of AR-positive cells and increased AR intensity in both epithelial and stromal cells in BPH compared to normal prostate.[24] However, the study of Hetzl et al. reported that the AR immunoreactivity in BPH was similar to that in normal prostate.[23] Naskar S et al stated that all the prostatic growth lesions in their study were positive for AR expression with varying staining intensity. Only 11.78% of BPH cases, 50% of PIN cases and 100% of carcinoma cases showed strongly positive AR expression.[11]

When the correlation between ER and AR in malignant and benign cases were assessed it was found that there was no correlation seen in malignant cases, however strong correlation was seen between AR and ER staining in benign cases. This correlation states that both androgen and estrogen receptors plays a crucial role in the progression of BPH.

5. Conclusions

Hormones play a multifactorial role in the pathogenesis of BPH and prostatic carcinoma; most commonly androgen and estrogen, and can be used towards finding a better therapy. Estrogen receptors are present only in small portion of prostatic tissue and with lesser intensity which was concordance with the study. AR expression is constantly present in benign and malignant prostatic epithelium. AR plays an important central role in growth and proliferation of prostate tissue so is a central focus for prostate carcinoma therapeutics. Our current and evolving understanding of AR with its positivity will hopefully shed light on selective treatment targets. However this study was not statistically significant as the sample size is small.

6. Conflict of Interest

The authors declare that they have no conflict of interest.

7. Source of Funding

None.

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