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Prognosis of Primary Breast Carcinoma with Expression of Androgen Receptor

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ABSTRACT

Background; Breast carcinoma is a heterogeneous malignancy with variable biological behavior and prognosis. Androgen receptor (AR) expression has recently emerged as a potential prognostic biomarker and therapeutic target in breast carcinoma. The present study was conducted to evaluate AR expression and its association with clinicopathological parameters, hormone receptor status, Ki-67 proliferative index, and prognosis in primary invasive breast carcinoma.

Materials and Methods; This cross-sectional study was conducted in the Department of Pathology at Believers Church Medical College Hospital from 2023 to 2024. A total of 50 cases of biopsy-proven primary invasive breast carcinoma were included. Immunohistochemistry was performed for AR, ER, PR, HER2/neu, Ki-67, and E-cadherin on formalin-fixed paraffin-embedded tissue sections. AR, ER, and PR were assessed using the Allred scoring system. Statistical analysis included Chi-square test, Mann–Whitney U test, Kruskal–Wallis test, correlation analysis, Kaplan–Meier survival analysis, and Cox proportional hazards regression analysis.

Results; AR positivity was observed in 66% of cases. A statistically significant positive correlation was found between age and AR expression ($p = 0.003$). AR expression demonstrated a significant inverse association with histological grade ($p = 0.001$) and Ki-67 proliferation index ($\rho = –0.53$, $p < 0.001$). A significant association was observed between AR positivity and ER positivity ($p = 0.004$). Triple-negative breast carcinomas showed significantly reduced AR expression ($p = 0.001$). Multivariate Cox regression analysis identified AR positivity as an independent favorable prognostic factor (adjusted HR = 0.25; $p = 0.041$).

Conclusion; Androgen receptor expression is associated with favorable clinicopathological characteristics, lower proliferative activity, hormone receptor positivity, and improved prognosis in primary breast carcinoma. AR may serve as an important prognostic biomarker and potential therapeutic target in breast carcinoma.

Keywords*: Breast carcinoma, androgen receptor, prognosis, immunohistochemistry, Ki-67, triple-negative breast carcinoma.*

INTRODUCTION

Breast carcinoma is the most frequently diagnosed malignancy among women worldwide and represents a major public health burden due to its increasing incidence and mortality.[1] According to GLOBOCAN 2020 estimates, breast cancer accounted for approximately 2.3 million new cases and 685,000 deaths globally, making it the leading cancer among women.[1] In India, breast carcinoma has surpassed cervical cancer as the most common female malignancy and contributes significantly to cancer-related morbidity and mortality.[2]

Breast carcinoma is a biologically heterogeneous disease characterized by considerable variability in morphology, molecular profile, clinical behavior, therapeutic response, and prognosis.[3] Histopathological evaluation and immunohistochemical profiling form the cornerstone of diagnosis, prognostication, and therapeutic planning in breast

cancer management.[4] Traditionally, breast carcinomas are categorized based on the expression of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor receptor 2 (HER2/neu), which have important predictive and prognostic implications.[5]

ER-positive and PR-positive breast cancers are generally associated with favorable prognosis and responsiveness to endocrine therapy.[6] HER2-positive tumors exhibit aggressive biological behavior but may respond effectively to HER2-targeted therapies such as trastuzumab.[7] Triple-negative breast carcinoma (TNBC), characterized by the absence of ER, PR, and HER2 expression, constitutes a highly aggressive subgroup associated with higher histological grade, increased proliferative activity, early recurrence, and poor survival outcomes.[8]

In recent years, the androgen receptor (AR) has emerged as an important biomarker in breast carcinoma. AR is a ligand-dependent nuclear transcription factor belonging to the steroid hormone receptor family and is normally expressed in breast epithelial cells.[9] Although androgen signaling has traditionally been studied in prostate carcinoma, increasing evidence suggests that AR also plays a significant role in breast carcinogenesis and tumor progression.[10]

AR expression has been reported in approximately 60–80% of invasive breast carcinomas, making it one of the most frequently expressed steroid receptors in breast cancer.[11] Several studies have demonstrated that AR-positive breast carcinomas are commonly associated with favorable clinicopathological characteristics such as lower histological grade, smaller tumor size, reduced proliferative index, hormone receptor positivity, and improved clinical outcomes.[12,13]

The exact biological role of AR in breast carcinoma remains complex and appears to vary according to molecular subtype.[14] In ER-positive tumors, AR signaling may exert anti-proliferative effects through competitive interaction with estrogen receptor pathways.[15] Conversely, in certain subsets of triple-negative breast carcinoma, AR signaling may promote tumor growth through alternative molecular pathways.[16]

Recent molecular profiling studies have identified a distinct “luminal androgen receptor” (LAR) subtype within triple-negative breast carcinoma, characterized by strong AR expression and unique transcriptional signatures.[17] This discovery has generated significant interest in AR as a potential therapeutic target, particularly in patients with TNBC who lack conventional hormone receptor targets.[18]

Ki-67 is an established marker of tumor proliferation and is widely utilized to assess biological aggressiveness in breast carcinoma.[19] Previous studies have shown an inverse association between AR expression and Ki-67 proliferative activity, suggesting that AR-positive tumors tend to exhibit lower proliferative potential and better prognosis.[20]

Despite growing international evidence regarding the prognostic significance of AR in breast carcinoma, data from the Indian population remain limited. Furthermore, the relationship between AR expression and clinicopathological characteristics, molecular subtype, proliferative activity, and prognosis continues to be an area of active investigation.

Therefore, the present study was undertaken to evaluate androgen receptor expression in primary invasive breast carcinoma and to analyze its association with clinicopathological parameters, ER, PR, HER2/neu status, Ki-67 proliferative index, and prognostic outcome.

MATERIAL AND METHODS

Study Design and Setting

This cross-sectional study was conducted in the Department of Pathology at Believers Church Medical College Hospital over a period from 2023 to 2024. The study aimed to evaluate androgen receptor (AR) expression and its association with clinicopathological parameters, hormone receptor status, HER2/neu expression, Ki-67 proliferation index, and prognosis in patients with primary invasive breast carcinoma.

Study Population

A total of 50 cases of biopsy-proven primary invasive breast carcinoma were included in the study. Female patients diagnosed with primary breast carcinoma and having adequate paraffin-embedded tissue blocks available for immunohistochemical analysis were enrolled.

Inclusion Criteria

1. All female patients with biopsy-proven primary breast carcinoma.
2. Availability of formalin-fixed paraffin-embedded tissue blocks suitable for immunohistochemistry.

Exclusion Criteria

1. Recurrent breast carcinoma cases.
2. Patients who had received prior chemotherapy or radiotherapy.

Sample Size
The minimum required sample size was calculated to be 48 cases, based on a prior study, with the following assumptions: Type I error of 5%, power of 80%, hazard ratio for survival prediction of 0.135, recruitment period of 2 years, minimum follow-up duration of 1 year, AR-positive to AR-negative ratio of 3:1, and median survival time of 10 years for AR-negative patients. A total of 50 cases were finally included in the study.

Data Collection
Patient demographic details including age and gender, along with clinical presentation and gross pathological characteristics such as tumor size and anatomical location, were retrieved from hospital medical records.

Histopathological Processing
The study was performed on paraffin-embedded tissue blocks obtained from tru-cut biopsies, wide local excisions, and mastectomy specimens. All specimens were fixed in 10% neutral buffered formalin. Representative tissue sections were selected and processed using an automated tissue processor, followed by paraffin embedding.

From each formalin-fixed paraffin-embedded tissue block, 4 µm thick sections were cut and stained with hematoxylin and eosin (H&E) for routine histopathological examination.

Immunohistochemistry
Immunohistochemical analysis was carried out for estrogen receptor (ER), progesterone receptor (PR), androgen receptor (AR), HER2/neu, E-cadherin, and Ki-67. The EP120 clone antibody was used for AR assessment. Prostatic tissue served as the positive control for AR immunostaining.

Immunohistochemistry Procedure
1. Four-micron-thick tissue sections were cut and mounted on charged slides.
2. Slides were incubated at 60–70°C for 1 hour.
3. Sections were deparaffinized using two changes of xylene for 5 minutes each.
4. Hydration was performed through descending grades of alcohol.
5. Antigen retrieval was carried out in MERS for 15 minutes.
6. Sections were washed in distilled water in three changes for 3 minutes each.
7. Slides were rinsed with PBS/TBS buffer for 2 minutes.
8. Endogenous peroxidase activity was blocked using hydrogen peroxide (H_2O_2) for 10 minutes.
9. Sections were washed twice with buffer for 2 minutes each.
10. Primary antibody against androgen receptor (EP120 clone) was applied and incubated for 45 minutes in a moist chamber.
11. Slides were washed with buffer three times for 2 minutes each.
12. Secondary antibody (Polyexcel Target Binder and Polyexcel HRP) was added and incubated for 10–12 minutes each.
13. Sections were again washed with buffer three times for 2 minutes each.
14. Diaminobenzidine (DAB) chromogen was applied, followed by washing in distilled water.
15. Counterstaining was performed using hematoxylin for 30 seconds.
16. Sections were dehydrated through ascending grades of alcohol, cleared in xylene, and mounted.

Evaluation of Immunohistochemistry
ER, PR, and AR Assessment
ER, PR, and AR expression were evaluated using the Allred scoring system, which combines the proportion score and intensity score.

The proportion score was assigned according to the percentage of positively stained tumour cells:

Percentage of Positive Cells	Proportion Score
0%	0
<1%	1
1–10%	2
11–33%	3
34–66%	4
>66%	5

The intensity of staining was graded as follows:

Intensity of Staining	Intensity Score
None	0
Weak	1
Intermediate	2
Strong	3

The final Allred score was calculated as the sum of the proportion and intensity scores. A total score of 0–2 was considered negative, while a score of 3–8 was regarded as positive.

HER2/neu Evaluation

HER2/neu expression was assessed according to the American Society of Clinical Oncology/College of American Pathologists (ASCO/CAP) guidelines and scored as 0, 1+, 2+, or 3+.

Score Interpretation

0 Faint/incomplete staining in ≤10% tumor cells

1+ Faint/incomplete staining in >10% tumor cells

2+ Weak/moderate complete staining in >10% tumor cells or strong complete staining in ≤10% tumor cells

3+ Strong complete staining in >10% tumor cells

Cases with equivocal HER2 expression (2+) underwent further evaluation using Fluorescence In Situ Hybridization (FISH). Tumors demonstrating HER2 gene amplification on FISH were classified as HER2 FISH-positive, whereas those lacking amplification were categorized as HER2 FISH-negative.

Ki-67 Proliferation Index

Ki-67 proliferation index was determined by calculating the percentage of tumour cells showing nuclear positivity. Tumours with Ki-67 expression <14% were considered to have a low proliferation index, whereas tumours with Ki-67 expression ≥14% were categorised as having a high proliferation index.

Follow-up and Prognostic Assessment

Prognosis was assessed by evaluating disease-free survival and overall mortality over a follow-up period of three years.

Statistical Analysis

Descriptive statistics were expressed as proportions and percentages. The association between androgen receptor expression and hormone receptor status was analysed using the Chi-square test. Odds ratios with 95% confidence intervals were calculated. Three-year survival rates were estimated using Kaplan–Meier survival curves, and differences between survival rates were compared using the log-rank test.

OBSERVATIONS AND RESULTS

A total of 50 cases of invasive breast carcinoma in female patients were studied in the Department of Pathology at Believers Church Medical College Hospital during the study period from January 2023 to December 2024. All cases fulfilled the predefined inclusion and exclusion criteria.

The mean age of the study population was 59.32 years, with an age range of 40–86 years. Age distribution showed normality on Shapiro–Wilk testing ($p = 0.503$).

Clinicopathological Characteristics of the Study Population

Table 1: Clinicopathological Characteristics of Patients with Primary Breast Carcinoma (n = 50)

Parameter	Category	Frequency (n)	Percentage (%)
Clinical Presentation	**Breast lump alone**	**37**	**74**
	Lump + nipple retraction + peau d'orange	**5**	**10**
	Lump + pain	**4**	**8**
	Lump + peau d'orange	**2**	**4**
	Lump + ulceration	**2**	**4**
Menopausal Status	**Premenopausal**	**7**	**14**
	Perimenopausal	**3**	**6**
	Postmenopausal	**40**	**80**
Family History	**Absent**	**45**	**90**
	Present	**5**	**10**
Tumor Laterality	**Right breast**	**28**	**56**

	Left breast	**22**	**44**
Tumor Focality	**Unifocal**	**44**	**88**
	Multifocal	**6**	**12**
Tumor Size	**≤2 cm**	**11**	**22**
	>2–5 cm	**33**	**66**
	>5 cm	**6**	**12**
Procedure Performed	**Core needle biopsy**	**46**	**92**
	Wide local excision	**3**	**6**
	Modified radical mastectomy	**1**	**2**
Tumor Location	**Retroareolar**	**6**	**12**
	Upper outer quadrant	**21**	**42**
	Lower outer quadrant	**8**	**16**
	Upper inner quadrant	**7**	**14**
	Lower inner quadrant	**8**	**16**
Histological Type	**Invasive breast carcinoma NST**	**44**	**88**
	Lobular carcinoma	**3**	**6**
	Mucinous carcinoma	**3**	**6**
Lymphovascular Invasion	**Absent**	**47**	**94**
	Present	**3**	**6**
Associated DCIS	**Absent**	**36**	**72**
	Present	**14**	**28**
Tumor Calcification	**Absent**	**42**	**84**
	Present	**8**	**16**
Tumor Infiltrating Lymphocytes	**Absent**	**22**	**44**
	Present	**28**	**56**
MBR Histological Grade	**Grade 1**	**7**	**14**
	Grade 2	**32**	**64**
	Grade 3	**11**	**22**

The most common presenting symptom was a breast lump alone, observed in 74% of cases. The majority of patients were postmenopausal (80%). Right-sided tumours were slightly more frequent (56%) than left-sided tumours (44%). Most tumours were unifocal (88%) and measured between >2 cm and 5 cm (66%). The upper outer quadrant was the most commonly involved site (42%).

Histologically, invasive breast carcinoma of no special type (IBC-NST) was the predominant subtype (88%). Lymphovascular invasion was identified in only 6% of cases, while associated ductal carcinoma in situ (DCIS) was observed in 28% of tumours. Tumour-infiltrating lymphocytes (TILs) were present in 56% of cases. Grade 2 tumours constituted the majority (64%) according to the Modified Bloom–Richardson (MBR) grading system.

Immunohistochemical Profile

Table 2: Immunohistochemical Profile of Breast Carcinoma Cases (n = 50)

Marker	Category	Frequency (n)	Percentage (%)
AR Status	**Positive**	**33**	**66**
	Negative	**17**	**34**
ER Status	**Positive**	**35**	**70**
	Negative	**15**	**30**
PR Status	**Positive**	**29**	**58**
	Negative	**21**	**42**
HER2/neu Status	**Negative**	**41**	**82**
	Equivocal	**4**	**8**
	Positive	**5**	**10**
Ki-67 Proliferative Index	**Low (<14%)**	**2**	**4**
	High (≥14%)	**48**	**96**
E-cadherin Expression	**Positive**	**45**	**90**
	Negative	**5**	**10**

AR positivity was observed in 66% of tumours. ER and PR positivity were identified in 70% and 58% of cases, respectively. HER2/neu overexpression was noted in 10% of tumours, while 8% showed equivocal HER2 expression.

A high Ki-67 proliferative index (≥14%) was observed in 96% of tumours, indicating high proliferative activity in the majority of cases. E-cadherin expression was retained in 90% of tumours.

Androgen Receptor (AR) Score

Table 3: Descriptive Statistics for Total AR Score

Variable	N	Minimum	Maximum	Mean ± SD
Total AR Score	**50**	**0**	**8**	**4.38 ± 3.40**

The total AR score ranged from 0 to 8, with a mean score of 4.38 ± 3.40, indicating moderate variability in AR expression among tumours.

Correlation Between Age and AR Expression

Table 4: Correlation Between Age and AR Expression

Variables	Pearson Correlation (r)	p-value
Age vs AR Expression	**0.417**	**0.003***

***Statistically significant ($p < 0.05$)**

A statistically significant moderate positive correlation was observed between age and AR expression ($r = 0.417$, $p = 0.003$), suggesting that AR positivity increases with advancing age.

Association of AR Expression with Clinicopathological Parameters

Table 5: Association of AR Expression with Clinicopathological Variables

Variable	Median AR Score	p-value
Clinical presentation	**6.0**	**0.423**
Menopausal status	**—**	**0.376**
Family history	**6.0**	**0.239**
Tumor location	**6.0**	**0.321**
Tumor laterality	**6.0**	**0.670**
Tumour focality	**6.0 vs 0.0**	**0.188**
Lymphovascular invasion	**6.0**	**0.736**
Associated DCIS	**6.0**	**0.466**
Calcification	**5.5 vs 6.0**	**0.372**
Tumour-infiltrating lymphocytes	**6.0**	**0.678**

No statistically significant association was observed between AR expression and clinical presentation, menopausal status, family history, anatomical site, laterality, focality, lymphovascular invasion, DCIS, calcification, or tumor infiltrating lymphocytes.

Association Between AR Expression and Histological Grade

Table 6: Association of Total AR Expression with MBR Histological Grade

MBR Grade	Number of Cases	Median AR Score	IQR	p-value
Grade 1	**7**	**6.0**	**6.0–7.5**	**0.001***
Grade 2	**32**	**7.0**	**3.8–7.0**	
Grade 3	**11**	**0.0**	**0.0–0.0**	

***Statistically significant ($p < 0.05$)**

A statistically significant inverse relationship was observed between histological grade and AR expression. Grade 3 tumours demonstrated complete loss or markedly reduced AR expression compared to Grade 1 and Grade 2 tumours.

Correlation of AR Expression with Ki-67 Proliferative Index

Table 7: Correlation Between Total AR Score and Ki-67 Index

Statistical Parameter	Value
Spearman's rho (ρ)	**–0.53**
p-value	**<0.001***

***Statistically significant ($p < 0.05$)**

A significant moderate negative correlation was observed between AR expression and Ki-67 proliferation index, indicating that tumours with higher AR expression exhibit lower proliferative activity.

Association Between AR and Hormone Receptor Status

Table 8: Association Between AR and ER Status

AR Status	ER Positive	ER Negative	p-value
Positive	**28**	**5**	**0.004***
Negative	**7**	**10**	

Odds Ratio = 8.0 (95% CI: 2.17–33.73)
***Statistically significant (p < 0.05)**

A significant association was identified between AR positivity and ER positivity. AR-positive tumours were eight times more likely to be ER-positive.

Table 9: Association Between AR and PR Status

Variable	p-value	Odds Ratio
AR vs PR	**0.13**	**2.79**

No statistically significant association was observed between AR and PR status.

Table 10: Association Between AR and HER2 Status

Variable	p-value
AR vs HER2	**0.44**

No statistically significant relationship was observed between AR expression and HER2 status.

AR Expression and Triple-Negative Breast Carcinoma

Table 11: Association of AR Status with Triple Negative Phenotype

Phenotype	AR Negative n (%)	AR Positive n (%)	p-value
Triple Negative Breast Carcinoma	**9 (75%)**	**3 (25%)**	**0.001***
Non-Triple Negative	**8 (21%)**	**30 (79%)**	

***Statistically significant (p < 0.05)**

AR expression was significantly reduced in triple-negative breast carcinoma (TNBC). Most TNBC cases lacked AR expression, whereas non-TNBC tumours predominantly demonstrated AR positivity.

AR Expression and Prognosis

Table 12: Prognostic Significance of AR Expression

Clinical Outcome	Number of Cases	Median AR Score	IQR	p-value
Stable during follow-up	**41**	**6.0**	**4.0–8.0**	**0.514**
Metastasis	**9**	**5.0**	**0.0–7.0**	

Although patients with stable disease demonstrated relatively higher AR scores, the difference was not statistically significant.

Multivariate Analysis

Analysis	Hazard Ratio (HR)	95% CI	p-value
Univariate (AR+)	**0.29**	**0.08–1.01**	**—**
Multivariate (AR+)	**0.25**	**0.06–0.99**	**0.041***

***Statistically significant (p < 0.05)**

Multivariate Cox proportional hazards regression analysis demonstrated that AR positivity was an independent, favourable prognostic factor after adjustment for tumour size and histological grade. AR-positive tumours showed approximately 75% reduced risk of adverse clinical outcomes compared to AR-negative tumours.

DISCUSSION

The present study evaluated androgen receptor (AR) expression in primary invasive breast carcinoma and analyzed its association with clinicopathological parameters, hormone receptor status, proliferative activity, and prognosis. AR positivity was observed in 66% of cases, which is consistent with previously published studies reporting AR expression in approximately 60–80% of invasive breast carcinomas.[11,12]

The mean age of the study population was 59.32 years, and a statistically significant positive correlation was observed between age and AR expression (p = 0.003). Similar findings have been documented by Castellano et al., who demonstrated increased AR positivity among older and postmenopausal women.[13] The higher AR expression observed in elderly patients may reflect hormonal alterations associated with menopause and decreased estrogenic influence.[21]

In the present study, the majority of tumors were invasive breast carcinoma of no special type (IBC-NST), which is in accordance with the known epidemiological distribution of breast carcinoma subtypes.[22] Most tumors were classified as Grade 2 according to the Modified Bloom–Richardson grading system.

A statistically significant inverse association was identified between AR expression and histological grade (p = 0.001). Grade 3 tumors demonstrated markedly reduced or absent AR expression compared with Grade 1 and Grade 2 tumors.

Similar observations have been reported by Rakha et al. and Park et al., who demonstrated that loss of AR expression is associated with poorly differentiated and biologically aggressive tumors.[23,24] Reduced AR expression in high-grade tumors may indicate dedifferentiation and increased genomic instability.[25]

The present study demonstrated a statistically significant negative correlation between AR expression and Ki-67 proliferation index ($\rho = -0.53$, $p < 0.001$). AR-negative tumors showed significantly higher proliferative activity compared to AR-positive tumors. These findings are comparable to those reported by McNamara et al. and Collins et al., who observed lower Ki-67 expression in AR-positive breast carcinomas.[20,26]

Ki-67 is an established marker of cellular proliferation and tumor aggressiveness.[19] The inverse relationship between AR and Ki-67 observed in the present study suggests that AR signaling may exert anti-proliferative effects in breast carcinoma. This supports the hypothesis that AR-positive tumors tend to exhibit more indolent biological behavior and favorable prognosis.[27]

ER positivity was observed in 70% of cases, while PR positivity was identified in 58% of tumors. A statistically significant association was observed between AR positivity and ER positivity ($p = 0.004$). AR-positive tumors were eight times more likely to be ER-positive compared to AR-negative tumors. Similar findings have been reported by Niemeier et al. and Castellano et al., who demonstrated strong concordance between AR and ER expression.[13,28]

The close association between AR and ER positivity may be explained by shared hormonal regulatory pathways and luminal differentiation.[29] In ER-positive tumors, AR signaling may inhibit estrogen-mediated proliferation through competitive binding at estrogen-responsive genomic elements.[15]

No statistically significant association was observed between AR expression and PR status or HER2/neu status in the present study. Similar findings have been documented in previous studies.[30] However, some investigators have reported increased AR expression in HER2-enriched molecular subtypes, suggesting possible crosstalk between HER2 and AR signaling pathways.[31]

An important finding of the present study was the significant reduction of AR expression in triple-negative breast carcinoma (TNBC) ($p = 0.001$). Approximately 75% of TNBC cases lacked AR expression, whereas the majority of non-TNBC tumors demonstrated AR positivity. Similar observations have been reported by Lehmann et al. and Choi et al.[17,32]

TNBC is characterized by aggressive clinical behavior, high proliferative activity, early metastasis, and poor prognosis.[8] The reduced AR expression observed in TNBC further supports the association between AR negativity and aggressive tumor biology. Nevertheless, a subset of TNBC cases demonstrated AR positivity, corresponding to the luminal androgen receptor (LAR) molecular subtype described in previous molecular profiling studies.[17]

The identification of AR-positive TNBC has important therapeutic implications because these tumors may respond to anti-androgen therapies such as bicalutamide and enzalutamide.[33] Therefore, AR evaluation may contribute not only to prognostic assessment but also to future therapeutic stratification in selected breast cancer patients.

The present study also assessed the prognostic significance of AR expression. Although the difference in raw AR scores between stable and metastatic cases was not statistically significant, multivariate Cox proportional hazards regression analysis demonstrated that AR positivity was an independent favorable prognostic factor after adjustment for tumor size and histological grade (adjusted HR = 0.25; $p = 0.041$).

These findings are comparable to studies conducted by Bozovic-Spasojevic et al. and Qu et al., who reported improved survival outcomes among patients with AR-positive breast carcinoma.[34,35] AR-positive tumors demonstrated approximately 75% lower risk of adverse clinical outcomes in the present study, supporting the role of AR as an independent prognostic biomarker.

No significant association was observed between AR expression and tumor laterality, tumor focality, lymphovascular invasion, calcification, tumor infiltrating lymphocytes, or associated ductal carcinoma in situ (DCIS). Similar findings have been reported in previous literature, indicating that AR expression is primarily related to intrinsic molecular characteristics rather than anatomical or inflammatory tumor features.[36]

The present study has certain limitations. The relatively small sample size and limited duration of follow-up may have reduced the statistical power for detecting certain associations. Additionally, molecular subtyping using advanced genomic techniques was not performed. Larger multicentric studies with longer follow-up duration are required to further validate the prognostic and therapeutic significance of AR in breast carcinoma.

Overall, the findings of the present study support the growing evidence that AR expression is associated with favorable clinicopathological features, lower proliferative activity, hormone receptor positivity, and improved prognosis in primary breast carcinoma.

CONCLUSION

Androgen receptor (AR) expression was observed in the majority of primary invasive breast carcinomas and was significantly associated with favourable prognostic factors, including lower histological grade, lower Ki-67 proliferative index, and estrogen receptor positivity. AR expression was significantly reduced in triple-negative breast carcinoma, indicating its association with aggressive tumour behaviour. Multivariate analysis demonstrated AR positivity as an independent favourable prognostic factor. These findings suggest that AR may serve as a useful prognostic biomarker and potential therapeutic target in breast carcinoma.

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