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Research Article

PATHOLOGIC COMPLETE RESPONSE AFTER NEOADJUVANT CHEMOTHERAPY IN NODE-POSITIVE BREAST CANCER PATIENTS: REGIONAL ANALYSIS OF THE BREAST AND AXILLA

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ABSTRACT

Objective: Pathologic complete response (pCR) after neoadjuvant chemotherapy (NAC) is a strong prognostic marker in breast cancer and may guide de-escalation of axillary surgery. This study evaluates breast, axillary, and overall pCR rates in initially node-positive patients and examines clinicopathologic predictors of response.

Materials and Methods: We retrospectively reviewed 212 women with cytology-proven node-positive invasive breast cancer treated with NAC followed by surgery between February 2019 and July 2025. Breast pCR was defined as ypT0/Tis, axillary pCR as ypN0, and overall pCR as simultaneous clearance of invasive disease in both regions. Associations between pCR and clinical stage, tumor characteristics, and molecular subtypes were analyzed.

Results: Breast, axillary, and overall pCR rates were 32%, 61%, and 29%, respectively. Axillary pCR exceeded breast pCR across all subtypes ($p<0.001$). HR–/HER2+ tumors showed the highest overall pCR (72.7%), whereas HR+/HER2– tumors had the lowest (15.9%). HER2 positivity and Ki-67 $\geqslant$20% were significantly associated with higher response rates. Clinical T and N stage, focality, and multifocality were not significantly correlated with pCR. Axillary response was particularly high in HR-/HER2+ and HR-/HER2- subtypes.

Conclusion: Molecular subtype is the strongest determinant of NAC response in node-positive breast cancer. The markedly higher axillary pCR rates compared with breast pCR highlight the possibility of axillary de-escalation, especially in HER2-positive and triple-negative disease.

Keywords: Breast cancer, Neoadjuvant chemotherapy, Pathologic complete response

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INTRODUCTION

Breast cancer(BC) is a leading cancer diagnosis in women and constitutes a major global public health concern due to its substantial contribution to both morbidity and mortality(1). In case of locally advanced BC, neoadjuvant chemotherapy (NAC) is widely preferred to downstage tumors, enhance the surgical feasibility (2). Pathologic complete response (pCR), one of the most valuable prognostic indicators following NAC, shows a positive correlation with long-term survival when detected in both the breast and axilla (3,4).

Earlier studies have demonstrated higher pCR rates is in triple-negative and human epidermal growth factor receptor 2 (HER2)–positive tumors compared with hormone receptor (HR)–positive subtypes (3,5). It has been shown that the rates of breast and axillary pCR do not always demonstrate a complete parallelism. Axillary pCR may be achieved independently from breast pCR; this finding is particularly important in surgical planning, especially regarding the necessity of axillary dissection. Axillary pCR has been reported to be higher than breast pCR, and this difference also varies across molecular subtypes (5). Tumor biology plays a critical role in determining surgical approaches after NAC. Significant differences in pCR rates have been identified among molecular subtypes, and it has been reported that pCR rates may range between 2% and 68% depending on subtype (6). HR+/HER2– tumors generally have the lowest pCR rates, whereas HR–/HER2+ tumors can reach much higher rates when appropriate systemic therapy is administered. In particular, in HER2–positive patients, combined treatment with targeted agents like trastuzumab and pertuzumab markedly increases response rates (7,8).

In addition to subtype, certain clinical and tumor-related characteristics of the patient may also affect NAC response. However, findings in the literature are partly inconsistent, and these factors are generally not as strong as molecular subtype. Numerous studies have investigated the effects of HER2 status, HR status, and proliferative activity (Ki-67) on NAC response (9,10,11). Tumor size and nodal burden may also influence the likelihood of response. Since NAC is typically administered to patients with larger tumors or multiple positive lymph nodes, these factors are expected to be negatively correlated with pCR (12).

In this context, our study aims to evaluate breast, axillary, and overall pCR rates in BC patients who initially presented with axillary lymph node positivity and received neoadjuvant chemotherapy; and to examine these responses in relation to molecular subtypes and clinical variables. This evaluation may provide guidance in determining the optimal surgical approach following treatment.

MATERIALS AND METHODS

Patients treated for primary invasive BC at Aydın Adnan Menderes University Hospital between February 1, 2019, and July 31, 2025, were retrospectively reviewed. Inclusion criteria comprised women aged ⩾18 years with clinically node-positive (cN+) invasive BC at diagnosis who received NAC followed by surgical treatment, corresponding to clinical stages cT1–4 and N1–3. Indications for NAC and treatment regimens were determined according to institutional protocols. Patients with locoregional recurrence or metastatic disease, those who underwent surgery prior to NAC, those treated with neoadjuvant endocrine therapy or neoadjuvant radiotherapy alone, and patients with inflammatory BC were excluded.

Electronic hospital records were examined to collect demographic, clinical, radiological, treatment, and pathological information. Clinical staging was performed according to the 7th edition of the American Joint Committee on Cancer (AJCC) tumor–node–metastasis (TNM) staging system (13). Patients with radiologically suspicious axillary lymph nodes confirmed by positive cytology were considered node-positive. Estrogen receptor (ER) and progesterone receptor (PR) status was considered positive when expression exceeded 1% of tumor cells. Ki-67 index was categorized into two groups: <20% or ⩾ 20%. HER2 status was assessed using immunohistochemistry (IHC) or fluorescence in situ hybridization (FISH), as appropriate.

Breast pCR was defined as the absence of invasive tumor in the breast (ypT0/Tis). Regarding axillary response, micrometastases were regarded as residual disease, whereas isolated tumor cells (ITCs) were regarded as of absence of residual disease. Axillary pCR was defined as ypN0/0i+. Overall pCR was defined as the simultaneous presence of both breast pCR (ypT0/Tis) and axillary pCR (ypN0/0i+).

Clinical and biological variables analyzed included age, prior to NAC clinical tumor size (cT stage), clinical nodal status (cN stage), multicentricity/focality, and receptor subtype (HR+/HER2–, HR+/HER2+, HR–/HER2+, and HR–/HER2–). Patients were categorized into three groups

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based on NAC response: breast pCR, axillary pCR, and overall pCR.

Surgical management consisted of breast-conserving surgery (BCS) or mastectomy, depending on multicentric disease, patient choice, and pathological findings. For axillary management after NAC, sentinel lymph node biopsy (SLNB) was performed in patients with cN0 disease; axillary lymph node dissection (ALND) was performed in cases with positive SLNB. In patients with cN-positive , axillary lymph node dissection (ALND) was performed.

Ethical Considerations

Ethical approval was obtained from the Non-Interventional Research Ethics Committee of Aydın Adnan Menderes University Faculty of Medicine (Approval No: 2025/357; 4 December 2025). The study adhered to the principles of the Declaration of Helsinki. Informed consent was waived due to the retrospective nature of the study.

Statistical Analysis

Statistical analyses were carried out using IBM SPSS Statistics version 26.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was examined with the Kolmogorov–Smirnov and Shapiro–Wilk tests. Data following a normal distribution were expressed as mean ± standard deviation, while non-normally distributed data were reported as median (range). Categorical variables were summarized as counts and percentages. Between-group comparisons were performed using the chi-square or Fisher's exact test for categorical data, and the independent t-test or Mann–Whitney U test for continuous variables, depending on data distribution. Statistical significance was defined as $p < 0.05$.

RESULTS

The study included 212 patients with a mean age of 54.3 ± 11.4 years. Of these, 40.1% were premenopausal and 59.1% were postmenopausal. The most common tumor location was the upper outer quadrant, identified in 54.2% of cases. Regarding tumor focality, 82.5% of patients had unifocal disease, 11.8% had multifocal tumors, and 5.7% presented with multicentric involvement. Histopathological evaluation showed that the majority of tumors (89.2%) were invasive ductal carcinoma (IDC). The distribution of clinical T stage was as follows: 25.9% T1, 59.4% T2, 9.9% T3, and 4.7% T4. Clinical nodal staging revealed that 42.5% were classified as N1, 44.8% as N2, and 12.7% as N3. HR positivity was present in 78.8% of patients, while HER2 positivity was identified in 38.7%. The Ki-67 proliferation index was <20% in 60.8% of cases and ⩾20% in 39.2%. Based on reseptor subtype classification, 50.5% of patients had HR+/HER2− subtype, 28.3% had HR+/HER2+, 10.8% had HR−/HER2+, and 10.4% had the HR−/HER2− (Table 1). A total of 141 patients (66.5%) underwent mastectomy, while 71 patients (33.5%) underwent BCS. The type of breast surgery differed significantly among reseptor subtypes ($p = 0.033$). The highest rate of BCS was observed in the HR−/HER2− subtype, in which 60.9% of patients underwent BCS and 39.1% underwent mastectomy. Regarding axillary surgery, 69.3% of patients underwent

Table 1. Baseline clinicopathologic characteristics of the patients

	n (%)
Age(years)	
Mean±SD	54.3±11.4
Menopausal status	
Premenopausal	85(40.1%)
Postmenopausal	127(59.1%)
Tumor Localization	
Upper Outer Quadrant	115(54.2%)
Upper Inner Quadrant	21(9.9%)
Lower Outer Quadrant	31(14.6%)
Lower Inner Quadrant	11(5.2%)
Central	34(16.0%)
Focality	
Unifocal	175(82.5%)
Multifocal	25(11.8%)
Multicentric	12(5.7%)
Histopathology	
IDC	189(89.2%)
ILC	10(4.7%)
Mucinous	5(2.4%)
Others	8(3.7%)
cT-stage	
T1	55(25.9%)
T2	126(59.4%)
T3	21(9.9%)
T4	10(4.7%)
cN-stage	
N1	90(42.5%)
N2	95(44.8%)
N3	27(12.7%)
HR Status	
Positive	167(78.8%)
Negative	45(21.2%)
HER2 Status	
Positive	82(38.7%)
Negative	130(61.3%)
Ki-67	
<20	129(60.8%)
≥20%	83(39.2%)
Reseptor Subtype	
HR+/HER2-	107(50.5%)
HR+/HER2+	60(28.3%)
HR-/HER2+	22(10.4%)
HR-/HER2-	23(10.8%)

SD, standard deviation; IDC, invasive ductal carcinoma; ILC, invasive lobular carcinoma; HR, hormone receptor; HER2, human epidermal growth factor receptor 2; Ki-67, Ki-67 proliferation index.

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Table 2. Surgical Approach and Sentinel Lymph Node Findings

Variables	All n (%)	HR+/HER2- n (%)	HR+/HER2+ n (%)	HR-/HER2+ n (%)	HR-/HER2- n (%)	*p*
Breast surgery						***0.033***
Mastectomy	141(66.5)	75(70.1)	42(70.0)	15(68.2)	9(39.1)	
BCS	71(33.5)	32(29.9)	18(30.0)	7(31.8)	14(60.9)	
Axillary Surgery						0.971
SLNB	147(69.3)	75(70.1)	42(70.0)	5(68.2)	15(65.2)	
ALND	65(30.7)	32(29.9)	18(30.0)	7(31.8)	8(34.8)	
Number of SLNs						0.170
Mean±SD	4.3±1.2	4.5±1.2	4.3±1.2	4.1±1.4	4.0±1.2	
Median(min-max)	4(2-6)	4(2-6)	4(2-6)	4(2-6)	4(2-6)	
SLNB Positivity						***0.018***
Positive	47(32.0)	32(42.7)	11(26.2)	3(20.0)	1(6.7)	
Negative	100(68.0)	43(57.3)	31(73.8)	12(80.0)	14(93.3)	
Non-SLN Involvement						0.718
Yes	24(51.1)	16(51.6)	6(50.0)	1(33.3)	1(100.0)	
No	23(48.9)	15(48.4)	6(50.0)	2(66.7)	0(0.0)	

HR, hormone receptor; HER2, human epidermal growth factor receptor 2; BCS, breast-conserving surgery; SLNB, sentinel lymph node biopsy; ALND, axillary lymph node dissection; SLN, sentinel lymph node; SD, standard deviation.

SLNB, whereas 30.7% underwent axillary ALND. The mean number of sentinel lymph nodes (SLNs) removed was 4.3 ± 1.2, with a median of 4 (2–6). Among patients who underwent SLNB, 32% had metastatic involvement in the sentinel node. The distribution of SLNB positivity differed significantly across molecular subtypes (p = 0.018). The lowest rate of SLN involvement was found in the HR−/HER2− subtype at 6.7%, whereas the highest rate was found in the HR+/HER2− subtype at 42.7%. Of the 47 patients with SLNB positivity, 24 (51.1%) had additional non-SLN metastases following ALND (Table 2).

In the entire cohort, breast pCR, axillary pCR, and overall pCR were observed in 32%, 61%, and 29% of patients, respectively. Among patients with HER2-positive tumor, breast, axillary, and overall pCR rates were markedly higher (p<0.001, p=0.044, and p<0.001, respectively). In contrast, patients with HR–positive tumors demonstrated significantly lower breast, axillary, and overall pCR rates (all p<0.001) (Table 3).

A marked difference in overall pCR distribution was observed among reseptor subtypes (p<0.001). Overall pCR rates were 15.9% in the HR+/HER2− subtypes, 35% in the HR+/HER2+, 72.7% in the HR−/HER2+, and 34.8% in the HR−/HER2−. Similarly, breast pCR rates differed significantly among subtypes (p<0.001). Breast pCR was achieved in 17.8% of patients with HR+/HER2− subtype, 35% of those with HR+/HER2+, 77.3% of those with HR−/HER2+, and 43.5% of those with HR−/HER2−. Axillary pCR rates also showed statistically significant variation according to reseptor subtype (p=0.001). Axillary pCR observed in 48.6% of patients with HR+/HER2− tumor, 65% of those with HR+/HER2+ tumor, 81.8% of those with HR−/HER2+ tumor, and 82.6% of those with HR−/HER2− tumor (Figure 1) (Table 3).

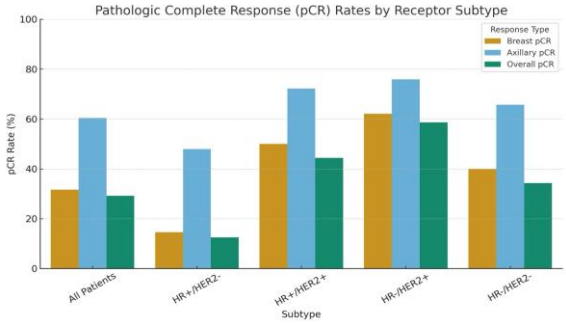

Figure 1. The figure illustrates breast, axillary, and overall pCR rates across receptor subtypes. Axillary pCR rates were consistently higher than breast pCR rates in all groups. The highest breast, axillary, and overall pCR rates were observed in the HR−/HER2+ subtype, followed by the HR−/HER2− subtype. In contrast, patients with HR+/HER2− tumors demonstrated the lowest pCR rates across all response categories. These findings highlight the strong association between tumor biology and treatment response following neoadjuvant chemotherapy.

DISCUSSION

NAC has taken on an increasingly prominent role in the management of BC and axillary disease. Achieving a pCR in the breast and axillary lymph nodes not only serves as a favorable prognostic indicator but also carries significant clinical value by enabling the use of less aggressive surgical approaches (3,4). In this study, breast, axillary, and overall pCR rates following NAC were evaluated separately in patients with initially node-positive BC, and these outcomes were analyzed in relation to clinicopathological characteristics and receptor subtypes. Our findings demonstrate marked differences in pCR rates

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Table 3. Comparison of breast, axillary, and overall pCR according to tumor characteristics.

	Breast			Axillary			Overall		
Variables	**Non-pCR n (%)**	**pCR n (%)**	***p***	**Non-pCR n (%)**	**pCR n (%)**	***p***	**Non-pCR n (%)**	**pCR n (%)**	***p***
All Patients	145(68.0)	67(32.0)	-	84(39)	128(61.0)	-	150(71.0)	62(29.0)	-
cT-stage			0.457			0.634			0.460
T1	37(67.3)	18(32.7)		21(38.2)	34(61.8)		39(70.9)	16(29.1)	
T2	86(68.3)	40(31.7)		52(41.3)	74(58.7)		89(70.6)	37(29.4)	
T3	13(61.9)	8(38.1)		6(28.6)	15(71.4)		13(61.9)	8(38.1)	
T4	9(90.0)	1(10.0)		5(50.0)	5(50.0)		9(90.0)	1(10.0)	
cN-stage			0.788			0.251			0.817
N1	63(70.0)	27(30.0)		30(33.3)	60(66.7)		63(70.0)	27(30.0)	
N2	65(68.4)	30(31.6)		43(45.3)	52(54.7)		69(72.6)	26(27.4)	
N3	17(63.0)	10(37.0)		11(40.7)	16(59.3)		18(66.7)	9(33.3)	
Focality			0.147			0.566			0.212
Unifocal	119(68.0)	56(32.0)		71(40.6)	104(59.4)		123(70.3)	52(29.7)	
Multifocal	15(60.0)	10(40.0)		10(40.0)	15(60.0)		16(64.0)	9(36.0)	
	11(91.7)	1(8.3)		3(25.0)	9(75.0)		11(91.7)	1(8.3)	
Multicentric									
HR Status			***<0.001***			***0.001***			***<0.001***
Positive	127(76.0)	40(24.0)		76(45.5)	91(54.5)		129(86.0)	38(61.3)	
Negative	18(40.0)	27(60.0)		8(17.8)	37(82.2)		21(14.0)	24(38.7)	
HER2 Status			***<0.001***			***0.044***			***<0.001***
Positive	44(53.7)	38(46.3)		25(30.5)	57(69.5)		45(54.9)	37(45.1)	
Negative	101(77.7)	29(22.3)		59(45.4)	71(54.6)		105(80.8)	25(19.2)	
Ki-67			***0.012***			0.121			***0.002***
<20%	97(75.2)	32(24.8)		57(44.2)	72(55.8)		102(79.1)	27(20.9)	
≥20%	48(57.8)	35(42.2)		27(32.5)	56(67.5)		48(57.8)	35(42.2)	
Subtype			***<0.001***			***0.001***			***<0.001***
HR+/HER2-	88(82.2)	19(17.8)		55(51.4)	52(48.6)		90(84.1)	17(15.9)	
HR+/HER2+	39(65.0)	21(35.0)		21(35.0)	39(65.0)		39(65.0)	21(35.0)	
HR-/HER2+	5(22.7)	17(77.3)		4(18.2)	18(81.8)		6(27.3)	16(72.7)	
HR-/HER2-	13(56.5)	10(43.5)		4(17.4)	19(82.6)		15(65.2)	8(34.8)	

cT, clinical tumor stage; cN, clinical nodal stage; pCR, pathological complete response; HR, hormone receptor; HER2, human epidermal growth factor receptor 2; Ki-67, Ki-67 proliferation index.

according to receptor status and show that axillary pCR rates were higher than breast pCR rates.

In clinically node-positive patients, the observed pCR rates were concordant with those reported in the literature, with breast pCR, axillary pCR, and overall pCR rates of 32%, 61%, and 29%, respectively (5,14,15). Biologically more aggressive subtypes, namely HR−/HER2+ and HR−/HER2− tumors, have been reported to achieve higher pCR rates in response to NAC (3,4,9). In line with these observations, our study demonstrated significantly higher breast, axillary, and overall pCR rates in HR−/HER2+ patients, reaching 77.3%, 81.8%, and 72.7%, respectively ($p<0.001$). In contrast, HR+/HER2− tumors, which rely predominantly on hormone receptor–mediated signaling, exhibited greater resistance to NAC, with breast, axillary, and overall pCR rates of 17.8%, 48.6%, and 15.9%, respectively. This reduced response is likely attributable to the lower chemosensitivity of these tumors and their dependence on estrogen receptor–driven proliferation (5,16). These findings are supported by systematic reviews confirming clinically meaningful differences in pCR rates among receptor-defined subtypes. Notably, a large-scale meta-analysis including more than 57,000 patients reported an overall pCR rate of approximately 13% in HR+/HER2− cases (17).

Overall pCR rates in HR−/HER2− tumors have generally been reported to range between 33% and 40% (3,4). However, some series have suggested that HR+/HER2+ tumors may achieve slightly higher pCR rates than HR−/HER2− tumors, similar to the findings observed in our cohort (9,17). In our study, the overall pCR rate was 35% in the HR+/HER2+ group and 34.8% in the HR−/HER2− group. Despite these comparable overall pCR rates, axillary response was more pronounced in the HR−/HER2− subgroup, with an axillary pCR rate of 82.6% compared with 65.0% in HR+/HER2+ patients. Differences in reported pCR rates across studies are often attributed to heterogeneity in pCR definitions or variations in neoadjuvant treatment regimens. In HER2-positive disease, hormone receptor status is known to play a critical role in treatment response, with HR+/HER2+ tumors demonstrating lower pCR rates compared with HR−/HER2+ tumors (9,18). Our findings are consistent with this established pattern and support the influence of hormone receptor expression on neoadjuvant treatment response.

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In our study, axillary pCR rates were significantly higher in the HR−/HER2− and HR−/HER2+ subtypes (82.6% and 81.8%, respectively) compared with other receptor-defined groups ($p<0.001$). Guo et al. reported an axillary pCR rate of 78% in HER2-positive and triple-negative patients with cN2 disease (19), while Cabıoğlu et al. similarly demonstrated an axillary response rate of 74% in patients who were initially node-positive (20). In patients achieving breast pCR, Lim et al. reported axillary pCR rates of approximately 88% in both HR−/HER2+ and HR−/HER2− subgroups (5). These findings suggest that axillary tumor cells may exhibit greater sensitivity to neoadjuvant therapy compared with the primary breast tumor. Given the consistently high axillary pCR rates observed in HR−/HER2− and HR−/HER2+ subtypes, several studies have explored the possibility of substantially reducing axillary surgery or even omitting it altogether in selected patients (20–23). These data support the concept that axillary surgical decision-making should be individualized based not only on the initial nodal stage but also on tumor biology and response to neoadjuvant therapy. Nevertheless, Samiei et al. reported axillary pCR rates among 544 cN1 patients with breast pCR (ypT0) of 31.9% in HR+/HER2−, 51.8% in HR+/HER2+, 46.7% in HR−/HER2+, and 48.5% in HR−/HER2− tumors, emphasizing that omission of axillary surgery should be approached with caution (24).

Among patients who became clinically node-negative after NAC and subsequently underwent SLNB, SLNB positivity was higher in hormone receptor–positive subtypes, whereas lower positivity rates were observed in HR−/HER2− and HR−/HER2+ patients. In our study, SLNB positivity was 42.7% in patients with HR+/HER2− disease, compared with 6.7% in the HR−/HER2− subgroup. These findings suggest that NAC can largely eradicate axillary disease, particularly in HR−/HER2− and HR−/HER2+ tumors, and support studies that have explored the de-escalation or omission of axillary surgery in selected patients. The relatively higher axillary pCR rates observed in our cohort may reflect differences in treatment eras as well as the impact of contemporary systemic and targeted therapies; therefore, these results should be interpreted cautiously.

Beyond receptor-defined subtypes, one of the most prominent clinicopathological factors associated with pCR is the tumor proliferation index (9,16,25). According to our findings, a Ki-67 value of $\geq$20% was markedly associated with higher breast and overall pCR rates, consistent with reports in the literature. In addition, hormone receptor negativity and HER2 positivity were also found to be associated with pCR. The relationship between cT and cN stage and pCR remains controversial in the literature. While some studies have reported that more advanced clinical stage is associated with lower pCR rates (26), others have found no significant association between clinical stage and pCR (16,27). In the GeparQuattro trial, although statistical significance was not reached, a trend toward higher pCR rates was reported in patients with cT1 tumors and node-negative disease (28). In our cohort, no statistically significant association was observed between either cT or cN and pCR. Nevertheless, smaller tumors appeared to achieve higher pCR rates; in our series, pCR was observed in 21.5% of cT1 tumors compared with approximately 8% of cT3 tumors. A similar trend has been reported in other studies (27). The impact of multifocality and multicentricity on pCR has been less extensively investigated. In our data,patients with multicentric tumors tended to have lower pCR rates, but this association did not achieve statistical significance. This result is in line with the expectation that a more extensive and dispersed tumor burden in multifocal or multicentric tumors may be more difficult to eradicate with neoadjuvant therapy. Supporting this hypothesis, Tinterri et al. demonstrated that among patients achieving pCR, those with residual in situ disease more frequently had multifocal or multicentric tumors at initial diagnosis (29).

The study has inherent limitations, primarily related to its retrospective methodology and single-center data source, which may constrain the broader applicability of the findings. Second, the relatively a limited number of cases across some subgroups restricted the ability to perform more detailed statistical comparisons between molecular subtypes. In addition, changes in neoadjuvant chemotherapy regimens and targeted therapies over time may have introduced treatment heterogeneity, potentially influencing response rates. Finally, long-term oncologic outcomes were not evaluated; therefore, conclusions regarding the prognostic impact of pCR remain limited.

CONCLUSION

In summary, the findings of this study indicate that reseptor subtypes play a considerable role in determining response to NAC and that axillary pCR rates are higher than breast pCR rates. Higher pCR rates were particularly demonstrated in patients with HER2-positive and triple-negative subtypes, indicating that these groups derive the greatest benefit from NAC. These findings provide important clinical insights for treatment planning, axillary surgical de-escalation, and the development of individualized surgical strategies. Taken together, the results highlight the need for prospective studies to more

precisely identify patient populations with favorable axillary response in whom axillary surgery can be safely reduced without compromising oncologic outcomes.

Acknowledgments
The authors deny any conflicts of interest related to this study

Authorship contributions
First Author: Concept, Design, Data Collection and Processing,Analysis, Literature Search, Writing; Second Author: Design, Data Collection and Processing, Literature Search

Data availibity statement
The data that support the findings of this study are available on request from the corresponding author.

Declaration of competing interest
The authors have no relevant financial or non-financial interests to disclose.

Ethics
This study was approved by the Ethics Committee for Noninvasive Clinical Trials of Aydın Adnan Menderes University. Approval date and number 2025/357.

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