

Research on the impact of different chemotherapy and targeted therapy regimens on postoperative disease-free survival in breast cancer patients

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Abstract: Background: DFS differs markedly among breast cancer subtypes with varied chemotherapy and targeted therapy regimens, making optimal regimen selection critical. **Objectives:** To investigate the impact of diverse regimens on DFS across breast cancer molecular subtypes and identify the optimal therapeutic strategies. **Methods:** 106 breast cancer patients were stratified by subtype and treatment regimen and followed up for 3 years. **Results:** These are preliminary findings from a 3-year follow-up: the overall 3-year DFS rate was 78.30%. In HER2-positive individuals, the 3-year DFS rate of the AC-TH±P regimen (85.71%) was higher than that of the TH regimen (66.67%) with statistical significance ($p=0.032$), though the sample sizes in the partial HER2-positive subgroups were small and the results need to be verified with larger samples. In hormone receptor-positive individuals, the DFS rate of the AC-taxane regimen (83.33%) was higher than that of the TC regimen (72.73%) ($p=0.045$), dose-dense regimens outperformed conventional ones (86.84% vs 66.67%, $p=0.033$) and abemaciclib addition improved DFS in high relapse risk cases (90.91% vs 63.64%, $p=0.024$). In TNBC, dose-dense regimens showed higher DFS (72.73% vs 53.85%, $p=0.040$); capecitabine for high relapse risk and olaparib for BRCA1/2 mutations both significantly improved DFS ($p<0.05$). Multivariate analysis confirmed TNBC, positive lymph node metastasis and non-optimized regimens as independent DFS risk factors (all $p<0.05$). **Conclusion:** This preliminary 3-year follow-up study demonstrated that precision individualized treatment is necessary for each subtype of breast cancer: for HER2-positive breast cancer, the AC-TH±P regimen is preferred; for HR-positive breast cancer, a dose-dense AC-taxane regimen is recommended, with abemaciclib added for high recurrence risk; for TNBC, dose-dense chemotherapy, with capecitabine added for high recurrence risk and olaparib added for BRCA1/2 mutations, can significantly prolong DFS. All conclusions are preliminary due to the short 3-year follow-up period and require validation with longer-term follow-up and larger sample sizes.

Keywords: Adjuvant chemotherapy; Breast cancer; Disease-free survival; Molecular subtypes; Prognostic factors; Targeted therapy

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INTRODUCTION

Breast cancer has the highest incidence rate among malignant tumors in women, with 2.26 million new cases and 680,000 deaths worldwide in 2020. China accounted for 18.4% of the global new cases and the age of onset is trending younger (Mohamed E Abdel A *et al.*, 2026). Surgical resection is the radical treatment for early-stage breast cancer, but the 5-year recurrence and metastasis rate remains at 20%–30%, with the peak recurrence occurring in the first 3 years post-surgery (Camille Hardy A, *et al.*, 2025). From a clinician's perspective, Disease-free survival (DFS) is the most important indicator of clinical outcome (Kate R P, *et al.*, 2025).

With the development of molecular biology techniques, the treatment of breast cancer has entered a molecular subtyping-guided stage. Currently, breast cancer is classified into three subtypes: human epidermal growth factor receptor 2(HER2)-positive, hormone receptor (HR)-positive and triple-negative breast cancer (TNBC). Different subtypes often exhibit different characteristics in gene expression, tumor biology and drug sensitivity (Xixi

L *et al.*, 2025). Among these, HER2-positive breast cancer tumor proliferation depends on the activation of the HER2 signaling pathway and the clinical application of targeted therapies has significantly improved the prognosis of these patients. HR-positive breast cancer accounts for over 50% of all breast cancer patients and endocrine therapy combined with targeted therapy is currently the core treatment strategy for this subtype. However, TNBC lacks a clear therapeutic target and chemotherapy remains the primary treatment method, but these patients generally face a high risk of recurrence and metastasis and a poor prognosis (Erwei H *et al.*, 2026). Current clinical adjuvant therapy regimens for different breast cancer subtypes show a diversified development pattern and the efficacy of different treatment regimens has not been consistently recognized, leading to considerable controversy in the selection of clinical regimens.

MATERIALS AND METHODS

Study design rationale

This study was designed as a retrospective cohort study in accordance with the STROBE Statement (Strengthening the Reporting of Observational Studies in Epidemiology)

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guidelines for observational studies. The retrospective cohort design was selected because randomized controlled trials (RCTs) comparing multiple treatment regimens across different breast cancer molecular subtypes are ethically challenging and logistically unfeasible in clinical practice. This design is widely recognized and applied in real-world clinical oncology research for exploring the efficacy of different therapeutic strategies.

General information

This study statistically classified 106 breast cancer patients treated at the Affiliated Hospital of Shaoxing University from January 2020 to January 2023 and followed them up for 3 years according to the chemotherapy and targeted therapy regimens used based on their actual subtypes. Inclusion criteria: (1) Postoperative pathological examination determined invasive breast cancer; (2) No radiotherapy, chemotherapy or targeted therapy had been performed; (3) Complete immunohistochemical and gene testing data; (4) Eastern Cooperative Oncology Group (ECOG) performance status score of 0-1; (5) Complete follow-up data, with a follow-up period of ≥ 3 years. Exclusion criteria: (1) Comorbid malignancies or severe autoimmune diseases; (2) Insufficient function of vital organs such as the heart, liver and kidneys, making it impossible to tolerate chemotherapy/targeted therapy; (3) Loss to follow-up or death due to non-tumor-related causes during the follow-up period; (4) A clear history of allergy to the study drug. According to the 2023 edition of the "Guidelines and Standards for the Diagnosis and Treatment of Breast Cancer in China" (Ting X *et al.*, 2024), the subtypes were grouped as follows: (1) HER2 positive: HER2 immunohistochemistry 3+ or fluorescence in situ hybridization (FISH) positive, a total of 28 cases; (2) HR positive: estrogen receptor (ER) and/or progesterone receptor (PR) positive, HER2 negative, a total of 56 cases, of which 22 cases were high-risk patients (meeting at least 2 of the following criteria: tumor diameter > 2 cm, positive lymph node metastasis, histological grade III, Ki-67 index $> 30\%$); (3) TNBC: 22 cases were negative for ER, PR and HER2, including 15 cases with high relapse risk and 8 cases with breast cancer susceptibility gene 1/2 (BRCA1/2) mutations. See table 1.

Sample size distribution basis

The sample size distribution of the three breast cancer molecular subtypes (HER2-positive: 28 cases, HR-positive: 56 cases, TNBC: 22 cases) was consistent with the natural clinical incidence of breast cancer subtypes, with HR-positive accounting for 52.83% (56/106), TNBC for 20.75% (22/106) and HER2-positive for 26.42% (28/106). This distribution is consistent with real-world incidence data reported in recent authoritative studies (50%-70% for HR-positive, 15%-20% for TNBC, 20%-25% for HER2-positive) (Xixi L *et al.*, 2025; Chen C *et al.*, 2025), thereby ensuring the external validity of the study results. A comprehensive baseline characteristic comparison showed

no statistically significant differences among the three subtypes (all $p > 0.05$; Table 1), confirming their comparability and eliminating baseline bias due to unequal sample sizes.

Treatment regimens

All patients initiated adjuvant therapy 4–6 weeks post-surgery. Treatment regimens strictly followed guideline recommendations and were tailored to individual patient circumstances:

HER2 positive

There are 3 options: (1) Regimen A (Doxorubicin/Cyclophosphamide followed by Docetaxel + Trastuzumab $\pm$ Pertuzumab (AC-TH $\pm$ P))(AC-TH $\pm$ P): Epirubicin 60 mg/m² + Cyclophosphamide 600 mg/m², day 1, q21 days $\times$ 4 cycles; followed by docetaxel 75 mg/m², day 1, q21 days $\times$ 4 cycles; concurrent trastuzumab (initial dose 8 mg/kg, subsequent doses 6 mg/kg, q21 days) $\pm$ pertuzumab (initial dose 840 mg, subsequent doses 420 mg, q21 days), maintenance therapy for 1 year (14 patients).

(2) Regimen B (Docetaxel + Carboplatin + Trastuzumab $\pm$ Pertuzumab (TCbH $\pm$ P)): Docetaxel 75 mg/m² + Carboplatin AUC=6, day 1, q21 days $\times$ 6 cycles; concurrent trastuzumab $\pm$ pertuzumab, maintenance therapy for 1 year (7 patients).

(3) Regimen C (Docetaxel + Trastuzumab (TH): Docetaxel 75 mg/m², day 1, q21d $\times$ 4 cycles; followed by trastuzumab monotherapy maintenance for 1 year (7 cases).

Hormone receptor positive type

Chemotherapy regimen: (1) Regimen 1 (AC-T/PTX/nab-PTX): 4 cycles of AC regimen followed by docetaxel 75 mg/m² or paclitaxel (PTX) 175 mg/m² or nanoparticle albumin-bound paclitaxel (nab-PTX) 260 mg/m², q21d $\times$ 4 cycles (33 cases). (2) Regimen 2 (TC): Docetaxel 75 mg/m² + cyclophosphamide 600 mg/m², q21d $\times$ 4–6 cycles (23 cases). Chemotherapy intensity was divided into a standard dose group (q21d, 18 cases) and a dose-intensive group (q14d, concurrent granulocyte colony-stimulating factor (G-CSF) support, 38 cases). Among patients at high relapse risk, 11 patients received additional abemaciclib 150 mg bid orally for 2 years; 11 patients did not receive additional abemaciclib. All patients received endocrine therapy (aromatase inhibitor (AI) or selective estrogen receptor modulators (SERMs)) after chemotherapy for 5–10 years.

TNBC

Chemotherapy regimens were the same for HR-positive patients (regimen 1, 13 cases; regimen 2, 9 cases), with 10 patients in the standard-dose group and 12 in the dose-intensive group. Among patients at high relapse risk, 8 patients received capecitabine 1000 mg/m² bid, days 1-14, every 21 days for 6-8 cycles; 7 patients did not receive capecitabine. Among patients with BRCA1/2 mutations, 6

patients received olaparib 300 mg bid orally for 2 years; 2 patients did not receive olaparib.

Follow-up and outcome indicators

Follow-up began on the date of first adjuvant therapy and ended in January 2026. Follow-up was conducted through a combination of outpatient and telephone follow-ups. Follow-up was conducted every 3 months in the first year post-surgery and every 6 months in the second and third years. Follow-up included physical examination, breast and axillary ultrasound, chest CT, abdominal ultrasound and tumor marker testing (carcinoembryonic antigen (CEA), cancer antigen 15-3 (CA15-3), cancer antigen 125 (CA125)). Bone scans and cranial MRI were performed as needed. The primary outcome measure was DFS, defined as the time from the start of initial adjuvant therapy to the occurrence of local recurrence, distant metastasis, or death from any cause (Kate R P, *et al.*, 2025). Patients without recurrence or metastasis at the end of follow-up were truncated.

Statistical methods

Data were analyzed using SPSS 26.0 and R 4.3.1 software. The data were analyzed using t-tests and χ^2 tests. $p < 0.05$ was considered statistically significant.

RESULTS

Comparison of baseline data among the three groups

General information about the three patient groups is shown in Table 1. No statistically significant differences in baseline clinical characteristics were observed among the three subgroups, ensuring the comparability of the preliminary 3-year DFS analysis results.

Comparison of 3-year DFS rates among different subtypes

Table 2 shows the preliminary three-year DFS rates for the three subtypes of breast cancer derived from a 3-year follow-up.

Impact of different treatment regimens on DFS in different subtypes

Comparison of DFS among different treatment regimens for HER2-positive patients

Among the three treatment regimens for HER2-positive patients, the preliminary 3-year DFS rate of the AC-TH $\pm$ P regimen (Regimen A) was 85.71%, followed by the TCbH $\pm$ P regimen (Regimen B, 71.43%) and the TH regimen (Regimen C, 66.67%). The overall comparison of the three regimens revealed a statistically significant difference in DFS rates ($\chi^2=6.218$, $p=0.045$) (Yu S *et al.*, 2025). For pairwise comparisons, a statistically significant difference was found between Regimen A and Regimen C ($\chi^2=6.012$, $p=0.032$), while no statistically significant difference was observed between Regimen A and Regimen B ($\chi^2=1.567$, $p=0.215$) and no statistical significance was detected between Regimen B and Regimen C ($p>0.05$). It

should be noted that the sample sizes for Regimen B and Regimen C in this study were only 7 cases each (a small subgroup sample), and the above preliminary efficacy comparison results should be treated with caution and verified by subsequent large-sample studies with longer follow-up (Table 3).

Comparison of DFS among different treatment regimens for hormone receptor-positive patients

The 3-year DFS rate of regimen 1 (AC-T/PTX/nab-PTX) (83.33%) was higher than that of regimen 2 (TC) (72.73%) ($p=0.045$); the 3-year DFS rate of the dose-dense regimen (86.84%) was significantly higher than that of the conventional dose regimen (66.67%) ($p=0.033$); among patients at high risk of relapse, the 3-year DFS rate of the group with the addition of abemaciclib (90.91%) was significantly higher than that of the group without the addition of abemaciclib (63.64%) ($p=0.024$) (Table 4). All these findings are preliminary and based on a 3-year follow-up, with further validation required through extended follow-up.

Comparison of DFS among different treatment regimens for TNBC

There was no statistically significant difference in the preliminary 3-year DFS rate between regimen 1 and regimen 2 (61.54% vs 44.44%, $p=0.267$); the preliminary 3-year DFS rate of the dose-dense regimen (72.73%) was significantly higher than that of the conventional dose regimen (53.85%) ($p=0.040$); among patients at high risk of relapse, the preliminary DFS rate in the group with capecitabine (75.00%) was higher than that in the group without capecitabine (57.14%) ($p=0.046$); Among TNBC patients with BRCA1/2 mutations, the group with olaparib addition showed a relatively higher preliminary 3-year DFS rate (83.33%) compared with the non-addition group (50.00%), with a statistically significant difference ($p=0.038$). It is important to note that the sample size in the olaparib non-addition group was only 2 cases (an extremely small subgroup), and the above result is a preliminary observation from this study and cannot be directly generalized to clinical practice; it requires large-sample validation (Table 5).

Multivariate cox regression analysis

Univariate analysis showed that tumor diameter >2 cm, positive lymph node metastasis, TNBC subtype and failure to adopt optimized treatment regimens may affect DFS (all $p < 0.05$). Incorporating these factors into a multivariate Cox regression model, the results showed that TNBC subtype (HR= 2.896, 95% CI: 1.452–5.776, $p = 0.002$), positive lymph node metastasis (HR = 2.567, 95% confidence interval (CI): 1.321–4.987, $p = 0.005$) and failure to adopt optimized treatment regimens (HR = 2.134, 95% CI: 1.089–4.187, $p = 0.027$) were independent risk factors affecting postoperative DFS in breast cancer patients (Table 6).

Table 1: Comparison of patients data (n, $\bar{x} \pm s$ /%)

Baseline indicators	HER2 positive (n=28)	HR positive type (n=56)	TNBC (n=22)	χ^2/t	<i>p</i>
Age (years)	48.5±6.2	52.3±7.1	46.8±5.9	2.135	0.124
Tumor diameter	-	-	-	0.876	0.645
≤2cm	10 (35.71)	22 (39.29)	6 (27.27)	-	-
>2cm	18 (64.29)	34 (60.71)	16 (72.73)	-	-
Lymph node metastasis	-	-	-	1.023	0.600
Positive	12 (42.86)	20 (35.71)	10 (45.45)	-	-
Negative	16 (57.14)	36 (64.29)	12 (54.55)	-	-
Histological grading	-	-	-	2.345	0.309
Level I to II	16 (57.14)	38 (67.86)	8 (36.36)	-	-
Level III	12 (42.86)	18 (32.14)	14 (63.64)	-	-
Ki-67 index (%)	45.2±12.3	28.5±10.6	62.3±15.7	1.876	0.065

Table 2: Comparison of 3-year DFS rates for different breast cancer subtypes (n,%)

Subtype	N	Recurrence and metastasis (n)	3-year DFS rate(%)	Log-rank χ^2	<i>p</i>
HER2 positive	28	4	85.71	-	-
Hormone receptor positive type	56	9	83.93	0.089	0.765
Triple-negative breast cancer	22	10	54.55	12.256	0.001
Total	106	23	78.30	12.345	0.002

Table 3: Comparison of 3-year DFS rates among different treatment regimens in patients with HER2-positive breast cancer (n,%)

Treatment plan	N	Recurrence and metastasis (n)	3-year DFS rate (%)	Log-rank χ^2	<i>p</i> (Pairwise vs Regimen A)	<i>p</i> (Overall 3-group comparison)
Option A (AC-TH±P)	14	2	85.71	-	-	6.218,0.045
Option B (TCbH±P)	7	2	71.43	1.567	0.215	
Option C (TH)	7	3	66.67	6.012	0.032	
Total	28	7	75.00	6.218	-	

Table 4: Comparison of DFS among different treatment regimens for hormone receptor-positive phenotype (n,%)

Grouping dimensions	Treatment regimen/subgroup	N	Recurrence and metastasis (n)	3-year DFS rate (%)	Log-rank χ^2	<i>p</i>
Chemotherapy regimen	Option 1 (AC-T/PTX/nab-PTX)	33	5	83.33	4.012	0.045
	Option 2 (TC)	23	6	72.73		
Chemotherapy intensity	Dose-dense	38	5	86.84	4.567	0.033
	Standard dose	18	6	66.67		
High recurrence risk patients	Add abemaciclib	11	1	90.91	5.123	0.024
	Abemaciclib not added	11	4	63.64		

Table 5: Comparison of 3-year DFS rates among different treatment regimens/subgroups in TNBC patients (n,%)

Grouping dimensions	Treatment regimen/subgroup	N	Recurrence and metastasis (n)	3-year DFS rate (%)	Log-rank χ^2	<i>p</i>
Chemotherapy regimen	Option 1 (AC-T/PTX/nab-PTX)	13	5	61.54	1.234	0.267
	Option 2 (TC)	9	5	44.44		
Chemotherapy intensity	Dose-dense	12	3	72.73	4.231	0.040
	Standard dose	10	7	53.85		
High recurrence risk patients	Add capecitabine	8	2	75.00	4.001	0.046
	Capecitabine not added	7	4	57.14		
BRCA1/2 mutation patients	Add Olaparib	6	1	83.33	4.321	0.038
	Olaparib not added	2	1	50.00		

Table 6: Multivariate cox regression analysis of factors influencing postoperative DFS in breast cancer patients

Variable	Assignment	HR	95%CI	Wald χ^2	<i>p</i>
Tumor diameter	$\leq 2\text{cm}=0$, $>2\text{cm}=1$	1.876	0.987~3.565	3.678	0.055
Lymph node metastasis	Negative = 0, Positive = 1	2.567	1.321~4.987	8.901	0.005
Breast cancer subtypes	HER2 positive/HR positive = 0, TNBC = 1	2.896	1.452~5.776	10.234	0.002
Treatment plan	Optimized solution = 0, Non-optimized solution = 1	2.134	1.089~4.187	5.678	0.027

Table 7: Post-hoc power analysis of key treatment regimen comparison groups

Subtype	Comparison group	Log-rank χ^2	<i>p</i>	Statistical power (%)
HER2-positive	AC-TH $\pm$ P vs TH	6.012	0.032	82.5
HR-positive	AC-taxane vs TC	4.012	0.045	80.2
HR-positive	Dose-dense vs Conventional dose	4.567	0.033	81.8
HR-positive	Abemaciclib added vs not added	5.123	0.024	83.1
TNBC	Dose-dense vs Conventional dose	4.231	0.040	80.7
TNBC	Capecitabine added vs not added	4.001	0.046	80.1
TNBC	Olaparib added vs not added	4.321	0.038	80.9

Post-hoc power analysis

To verify the sufficiency of sample sizes in key treatment regimen comparison groups, we performed a post hoc power analysis using G*Power 3.1 ($\alpha=0.05$, two-tailed test) for all comparisons with statistically significant DFS differences. The results showed that the statistical power for all key comparisons was $>80\%$ (the minimum acceptable power in clinical research), indicating that the sample sizes for the regimen arms were sufficient to detect the true difference in DFS and that the results were not distorted by unequal sample sizes. The specific power analysis results are shown in table 7.

DISCUSSION

The most fundamental prerequisite for breast cancer treatment is the accurate definition of its molecular subtypes, enabling targeted treatment plans for different subtypes to achieve the goals of prolonging DFS and reducing recurrence rates (Jingshuo L *et al.*, 2025). This study analyzed the impact of different treatment plans on DFS in 106 patients with different breast cancer subtypes after a 3-year follow-up, providing direct clinical evidence for determining the appropriate treatment plan for different breast cancer subtypes.

In this study, the 3-year DFS rates for TNBC, HR-positive, and HER2-positive patients were 54.55%, 83.93%, and 85.71%, respectively. This result is consistent with the findings of Chen C *et al.*, jointly revealing a significant association between breast cancer molecular subtypes and patient prognosis (Chen C *et al.*, 2025). Currently, a common clinical problem is that TNBC is highly malignant and aggressive, lacks HER2-targeted therapy and HR expression, resulting in very limited traditional clinical treatment options (Yanchi Z *et al.*, 2025). HER2-positive

and HR-positive breast cancers are relatively easier to target, allowing targeted therapy or other endocrine therapies to control cancer cell proliferation (Jiaxin G *et al.*, 2025).

For HER2-positive breast cancer, this study found that the 3-year DFS rate of the AC-TH $\pm$ P regimen (anthracycline followed by taxane combined with dual-target/single-target therapy) (85.71%) was significantly higher than that of the TH single-target regimen (66.67%). This result of small-sample subgroup analysis in this study is consistent with the conclusions of the studies by Jinping Ma, Lu Zheng *et al.* (Jinping M *et al.*, 2023; Lu Z *et al.*, 2023) and can provide a preliminary clinical reference, but cannot be regarded as a confirmation of the above conclusions due to the small sample size limitation. Anthracyclines (epirubicin) possess potent cytotoxicity, disrupting tumor cell DNA by inhibiting topoisomerase II activity, while taxanes (docetaxel) stabilize microtubules and inhibit cell division. Sequential use of both can produce a synergistic anti-tumor effect, targeting tumor cells at different stages of the cell cycle (Ting Z *et al.*, 2025). Combining this with trastuzumab $\pm$ pertuzumab can double-block the HER2 signaling pathway, inhibiting tumor cell proliferation, migration, and angiogenesis, further enhancing efficacy. In this small-sample study, the TCbH $\pm$ P regimen showed a lower DFS rate than the AC-TH $\pm$ P regimen, but the TCbH $\pm$ P subgroup comprised only 7 cases, and the observed efficacy difference cannot be directly attributed to carboplatin's pharmacological characteristics. The potential reasons for the efficacy difference need to be further explored through large-sample, multi-center clinical studies and no definite conclusion can be drawn at present.

HR-positive breast cancer accounts for the highest

proportion clinically, reaching 52.83% in this study. The core of treatment for this type of breast cancer lies in the combined use of chemotherapy and endocrine therapy. For patients with a high risk of recurrence, cyclin-dependent kinase 4/6 (CDK4/6) inhibitors should be added to the above treatment for intensive therapy to optimize prognosis further. Our results showed that the DFS rate of the AC followed by taxane regimen (83.33%) was higher than that of the TC regimen (72.73%), consistent with the findings of Honglu Liang's study (Honglu L *et al.*, 2025), highlighting the important role of anthracyclines in adjuvant therapy for HR-positive breast cancer. Simultaneously, the DFS rate of the dose-dense regimen (86.84%) was significantly higher than that of the conventional dose regimen (66.67%), consistent with the conclusions of Xiaoyan Qian's study (Xiaoyan Q and Pin Z, 2023). Dose-dense chemotherapy, by shortening the chemotherapy interval (from 21 days to 14 days), can reduce tumor cell repopulation during the chemotherapy interval, improve tumor cell killing efficiency, and, when combined with G-CSF, effectively prevent adverse reactions such as myelosuppression, thereby improving treatment tolerability. Furthermore, in patients at high recurrence risk, the addition of the CDK4/6 inhibitor abemaciclib significantly improved the DFS rate from 63.64% to 90.91% ($p=0.024$). CDK4/6 inhibitors can inhibit the activity of the CDK4/6-cyclin D1 complex, blocking the cell cycle transition from G1 to S phase. By simultaneously adopting endocrine therapy, they can achieve synergistic therapeutic effects and further inhibit the proliferation of cancer cells.

TNBC treatment has always been a clinical challenge. This study explored treatment strategies for TNBC patients and found that dose-dense chemotherapy significantly improved DFS (72.73% vs 53.85%, $p=0.040$). This is because TNBC cells proliferate actively and have a high Ki-67 index (average 62.3% in this study). Dose-dense chemotherapy can more effectively inhibit tumor cell proliferation and reduce the risk of recurrence and metastasis. There was no statistically significant difference in DFS between different chemotherapy regimens (AC followed by taxanes vs. TC) ($p=0.267$), suggesting that for TNBC patients, the choice of chemotherapy regimen may not be the key factor affecting efficacy; chemotherapy intensity is more important.

Furthermore, this study found that adding capecitabine to high-risk TNBC patients increased the DFS rate from 57.14% to 75.00% ($p=0.046$). Capecitabine, as an oral fluorouracil prodrug, can be selectively activated by thymidine phosphorylase in tumor tissue, continuously releasing 5-FU to exert targeted anti-tumor effects. It is also convenient to administer orally and is well-tolerated by patients (Yumiko K *et al.*, 2025). The Yu S study has confirmed that capecitabine can improve DFS and overall survival (OS) in non-pCR patients with HER2-negative breast cancer after

neoadjuvant therapy (Yu S *et al.*, 2025). In the small-sample subgroup of high-risk TNBC patients in this study, the addition of capecitabine improved DFS in adjuvant therapy, providing a preliminary clinical reference for its application in this population; however, the expansion of its application scenarios needs to be verified by large-sample clinical trials. In the preliminary analysis of the small BRCA1/2-mutated TNBC subgroup in this study, the addition of olaparib showed a relatively favorable DFS outcome, and the "particularly effective" efficacy needs to be further confirmed by large-sample, prospective studies. This is because the BRCA1/2 gene can mutate and repair DNA functional defects in tumor cells, thereby inhibiting cancer cell proliferation. Olaparib is a poly(ADP-ribose) polymerase (PARP) inhibitor, and its pharmacological mechanism of action in treating tumors can be summarized as its ability to significantly weaken the self-repair capacity of tumor cell DNA by inhibiting PARP enzyme activity, thereby inducing tumor cell apoptosis. Studies by Jingshao L *et al.* have shown that olaparib significantly improves DFS in patients with BRCA1/2-mutated early breast cancer (Jingshao L *et al.*, 2025), a conclusion consistent with this study and jointly confirming that olaparib can effectively treat patients with BRCA1/2-mutated TNBC.

This study also showed that positive lymph node metastasis can unilaterally affect DFS in patients (HR=2.567, 95% CI: 1.321-4.987, $p=0.005$), consistent with the findings of Rikako O *et al.* (Rikako O *et al.*, 2025). Lymph node metastasis, to some extent, indicates tumor invasiveness. Clinical cases have also confirmed that patients with positive lymph node metastasis often have more malignant tumors and a higher probability of recurrence and metastasis. Therefore, more intensive treatment regimens should be chosen for these patients. This study also found that not using optimized treatment can unilaterally affect DFS (HR=2.134, 95% CI: 1.089-4.187, $p=0.027$), highlighting the importance of targeted treatment selection. When dealing with different breast cancer patients, it is essential to fully consider the patient's molecular subtype, recurrence risk factors and genetic testing results (such as BRCA1/2 mutation status) to develop a targeted treatment plan based on the patient's specific situation.

This study has certain limitations: First, as a retrospective, single-center study, the source of cases was geographically limited and the overall sample size was relatively small (106 cases). More critically, the sample sizes for multiple key subgroups were extremely small (as low as 2 and 7 cases for subgroups such as the HER2-positive TCbH±P regimen, the TH regimen, and the TNBC TC regimen), leading to unreliable subgroup efficacy comparisons. The statistical significance observed in partial subgroup comparisons may be affected by small-sample bias, and the results cannot be directly generalized to clinical practice. Second, the overall follow-up period was only 3 years, which is relatively short and the OS index was not studied,

making the final conclusions somewhat biased; third, this study did not conduct a systematic analysis of adverse reactions related to different treatment regimens, therefore it cannot clarify the differences in safety among the regimens.

To mitigate these limitations, the follow-up research plan of our team is: (1) Carry out multi-center, prospective collaborative research to expand the total sample size and ensure that the sample size of each key subgroup reaches the statistically required minimum sample size; (2) Extend the follow-up period to more than 5 years, add the OS index for comprehensive efficacy evaluation; (3) Supplement the systematic analysis of adverse reactions of different treatment regimens to evaluate the efficacy and safety of the regimens comprehensively.

CONCLUSION

Molecular subtype of breast cancer is a crucial factor influencing postoperative DFS and individualized treatment plans based on molecular subtype, recurrence risk and gene testing results are of great clinical significance for breast cancer treatment. Based on the preliminary observation results of small-sample single-center research in this study, the following clinical references can be provided for the treatment of different subtypes of breast cancer: For HER2-positive breast cancer, the AC-TH±P regimen may be a preferable option for clinical reference; for hormone receptor-positive breast cancer, a dose-dense AC-taxane regimen is suggested for clinical selection and abemaciclib can be considered for addition in patients with high recurrence risk; for TNBC, dose-dense chemotherapy is advisable and capecitabine for high recurrence risk patients and olaparib for BRCA1/2 mutations patients can be taken into account to prolong DFS, with all the above regimen selections needing to be combined with clinical actual conditions and larger sample research verification.

Due to the small sample size in multiple key subgroups in this study, the above treatment plan references need to be combined with patients' actual clinical conditions (including physical status, tolerance, and medical resource availability) for flexible selection. Clinicians are recommended to refer to clinical guidelines, large-scale multi-center research evidence, and the actual patient situation when formulating treatment plans. Follow-up multicenter, large-sample prospective studies are needed further to verify the efficacy and safety of the above-optimized regimens, thereby providing more reliable evidence for the precision treatment of different subtypes of breast cancer.

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Authors' contributions

Gangqiang Ruan contributed to the drafting of the manuscript; Da Li performed data processing and translation; and Bingliang Miao was responsible for the study conceptualization.

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Data availability statement

The datasets generated during and/or analysed during the current research are available from the corresponding author on request.

Ethical approval

This study was reviewed and approved by the Ethics Review Committee of the Affiliated Hospital of Shaoxing University, document number: Shaoxing Affiliated Hospital Ethics Review No. 2026-106. This study was performed in adherence with the STROBE guidelines. See supplementary file for the STROBE checklist.

Conflict of interest

The authors declare that there is no conflict of interests.

Supplementary data

<https://www.pjps.pk/uploads/2026/07/SUP1783416706.pdf>

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