Pathological response to neoadjuvant chemotherapy and survival in patients with breast cancer: A multicentric retrospective study

EVRARD NARCISSE SÉKA[1,2], DANIELLE ASMAO TRAORÉ[3,4], CORNEILLE TÉA SAKI[2,5], KADJATOU TRAORÉ[4,6], AMARA GOZANA TUO[1] and BRAHIMA DOUKOURÉ[2,7]

[1]Department of Radiotherapy, Alassane Ouattara National Center for Medical Oncology and Radiotherapy, 08 BP 4104 Abidjan 08, Abidjan-Cocody, Republic of Côte d'Ivoire; [2]Medical Sciences Training and Research Unit, Félix Houphouet-Boigny University, 01 BP V34 Abidjan 01, Abidjan, Republic of Côte d'Ivoire; [3]Department of Medical Oncology, Bouaké Hospital and University Center, 01 BP 12885 Bouaké 01, Republic of Côte d'Ivoire; [4]Medical Sciences Training and Research Unit, Alassane Ouattara University, 01 BP V18 Bouaké 01, Republic of Côte d'Ivoire; [5]Department of Gynecology and Obstetrics, Treichville Hospital and University Center, 01 BP V3 Abidjan 01, Abidjan, Republic of Côte d'Ivoire; [6]Department of Medical Oncology, Alassane Ouattara National Center for Medical Oncology and Radiotherapy, 08 BP 4104 Abidjan 08, Abidjan-Cocody, Republic of Côte d'Ivoire; [7]Department of Pathology, Cocody Hospital and University Center, 01 BP V13 Abidjan 01, Abidjan, Republic of Côte d'Ivoire

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Abstract. In several European and American countries, pathological complete response (pCR) to neoadjuvant chemotherapy (NAC) in breast cancer has been identified as a predictor of improved survival. However, data from Black African patients are lacking. The present study aimed to evaluate the pathological response to NAC and its impact on survival in patients with breast cancer. A retrospective analysis of patients with breast cancer treated with NAC from January 2017 to December 2024 (96 months) was performed to assess associations between clinicopathological variables and pCR using a binary logistic regression test. Survival rates were determined using the Kaplan-Meier method. Survival comparisons in univariate analysis were performed using the log-rank test. A Cox regression model was used for multivariate analysis. The mean age of the 195 included patients was 46.3 years. The predominant histological type was invasive carcinoma of no special type (91.3%). Stage III tumors accounted for 69.8% of cases. The molecular subtypes were: Luminal (39.5%), HER2-positive (20.5%) and triple-negative (TN; 40.0%). NAC consisted primarily of sequential anthracycline-taxane regimens (94.4%). pCR was achieved in 28.7% of cases. HER2-positive tumors were significantly associated with pCR (odds ratio, 2.62; 95% CI, 1.10-6.22). Multivariate analysis revealed that pCR was the only factor significantly associated with both progression-free survival [PFS; hazard ratio (HR), 6.54] and overall survival (OS; HR, 5.84;. Within each molecular subtype, pCR was associated with improved PFS. In the TN group only, pCR was associated with improved OS. Pathological response was the strongest independent predictor of improved PFS and OS. HER2-positive tumors demonstrated the highest probability of pCR, while only the TN subtype showed a significant improvement in both OS and PFS with the achievement of pCR.

Correspondence to: Dr Evrard Narcisse Séka, Medical Sciences Training and Research Unit, Félix Houphouet-Boigny University, 82W4 + 3W4 Cocody University Boulevard, 01 BP V34 Abidjan 01, Abidjan, Republic of Côte d'Ivoire
E-mail: docsena2810@gmail.com

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Introduction

Historically, breast cancer management followed a standard sequence of surgery, followed by chemotherapy and radiotherapy (1). However, the use of chemotherapy before surgery, in a neoadjuvant setting, shows similar survival outcomes to adjuvant therapy (2), but it offers some clinical advantages. Initially, it was used to downstage and render operable locally advanced breast cancers that were inoperable from the outset, or to enable conservative surgery for certain operable, locally advanced forms (3). Indications of neoadjuvant chemotherapy (NAC) have extended to include early-stage triple-negative (TN) and HER2-positive subtypes, supported by the positive predictive value of pathological complete response (pCR) for survival (4,5). Studies have demonstrated that pathological responses to NAC vary between the intrinsic molecular subtypes (1,6,7). Pathological response following neoadjuvant chemotherapy was also used to guide adjuvant systemic. Patients who experience a pCR had a good prognosis, and did not benefit from adjuvant therapy (except endocrine therapy in endocrine receptor-positive breast cancer). HER2-positive and triple negative cancer patients with incomplete response would

benefit from additional adjuvant systemic therapy (8,9). Most of these studies come from developed countries, and thus, mostly include White patients (10,11). However, breast cancer presents racial and ethnic disparities (12). Therefore, the results of these studies cannot be generalized systematically to Black African patients. Breast cancer is a heterogeneous disease with molecular and phenotypical subtypes that vary across racial and ethnic groups (13). These subtypes influence prognosis and treatment outcomes (14). In the Republic of Côte d'Ivoire, breast cancer is the most prevalent cancer among female patients, with 3869 new cases in 2022 (33.5% of all new cancer cases) according to the Globocan statistics (15). From 2008 to 2015, most cases were diagnosed at advanced stages (nearly 65% at stages III or IV), often requiring the use of NAC (16). To the best of our knowledge, pCR following NAC and its impact on survival outcomes has not yet been studied. Therefore, the present study aimed to evaluate the pathological response to NAC and its influence on survival in patients with breast cancer.

Patients and methods

Patient selection and data collection. The present study was a retrospective analytical study of patients treated from January 2017 to December 2024. The present study included 238 female patients aged ≥18 years. Inclusion criteria were: Pathologically confirmed, non-metastatic breast carcinoma who were treated with NAC followed by surgery in three tertiary hospitals in the Republic of Côte d'Ivoire (Alassane Ouattara National Center of Medical Oncology and Radiotherapy of Abidjan, Treichville Hospital and University Center of Abidjan, and Bouaké Hospital and University Center, Bouaké); available pathological examination of the surgical specimen.

Description of the chemotherapy protocols, the number of cycles, and the subsequent treatments such as radiotherapy, endocrine therapy or adjuvant chemotherapy. The present study was approved by The National Ethical Committee of Life and Health Sciences (approval no. 00068/25/MSHPCMU/CNESVS-km) of Republic of Côte d'Ivoire. All patients gave their written consent to participate to the study. Pathological and immunohistochemistry analyses were performed on a breast core biopsy before the chemotherapy course. Computed tomography scans of the chest, abdomen and pelvis were performed to rule out distant metastasis. Pathological, immunohistochemistry and computed tomography scan data were obtained from the medical records of the patients. Tumor staging was based on the eighth edition of the American Joint Committee on Cancer/Union for International Cancer Control (UICC) system (17).

The exclusion criteria were as follows: Patients who received radiotherapy prior to surgery; patients with a delay >12 weeks between NAC and surgery; patients with a delay >6 weeks between cycles of chemotherapy; patients with a follow-up time <1 year; and patients lost to follow-up after the end of treatment for whom post-treatment data were unavailable.

The collected data included: Age; tumor stage and grade; estrogen receptor (ER) status; progesterone receptor (PR) status; HER2 status; molecular subtype; chemotherapy regimen; treatment following surgery (radiotherapy, hormonal therapy, anti-HER2 therapy or adjuvant chemotherapy); pathological response; and overall survival (OS) and progression-free survival (PFS).

Molecular subtypes were categorized as luminal, HER2-positive or TN based on ER, PR and HER2 status. The ER, PR and HER2 status was assessed by immunohistochemistry. Data were extracted from the immunohistochemistry exam reports in the patient medical records. ER and PR were considered positive if ≥10% of cells were stained positive. Evaluation of HER2 expression was based on the degree of membrane staining as follows: 0, no or incomplete, faint/barely perceptible membrane staining in ≤10% of invasive tumor cells; 1, incomplete, faint/barely perceptible membrane staining in >10% of invasive tumor cells; 2, incomplete and/or weak to moderate circumferential membrane staining in >10% of invasive tumor cells or complete, intense, circumferential membrane staining in ≤10% of invasive tumor cells

Score 3 corresponds to complete, intense, circumferential membrane staining in >10% of invasive tumor cells.

HER2 was considered upregulated (HER2-positive) if the score was 3; scores of 0 and 1 indicated an absence of HER2 upregulation. If the score was 2, an *in situ* hybridization test was required.

Tumors were classified as follows: Luminal, ER- and/or PR-positive, HER2-negative; HER2-positive, HER2-positive with or without ER or PR expression; and TN, ER-, PR- and HER2-negative.

pCR was defined as an absence of residual invasive or micro-invasive disease in both the breast (the primary tumor site) and axillary lymph nodes (ypT0/ypTis, ypN0 based on the 8th edition of breast cancer staging of the AJCC)(17). The pathological response was reported according to whether pCR was achieved [absence of complete response group (no pCR) and complete response group (pCR)].

PFS was defined as the time from diagnosis to recurrence (locoregional and/or distant) or death from any cause. OS was defined as the time from diagnosis until death from any cause.

Statistical analysis. Statistical analysis was performed using IBM SPSS Statistics version 27 (https://www.ibm.com/support/pages/downloading-ibm-spss-statistics-27). Baseline characteristics of patients, tumors and treatment were presented as mean ± SD) for continuous variables, or as proportions for categorical variables. Binary logistic regression analysis was performed to detect associations between clinicopathological variables (age, histological subtype, grade, molecular subtype and stage) and pathological response. χ^2 test was used for comparison. There was a significant association between the variable and pathological response if P-value was <0,05. Odd ratio (OR) and 95% confident interval were reported for each variable. Survival was assessed using the Kaplan-Meier method on an intention-to-treat basis. Univariate and multivariate analyses were performed to assess the influence of clinicopathological variables on PFS and OS. The log-rank test was used for the univariate analysis. Variables that were significant (P<0.05) in the univariate analysis were included in the multivariate analysis using a Cox regression model.

Table I. Patient and tumor characteristics.

Variable	Luminal (%)	HER2-positive (%)	Triple-negative n (%)	Total (%)
Age, years				
<40	16 (8.2)	15 (7.7)	21 (10.8)	52 (26.7)
≥40	61 (31.3)	25 (12.8)	57 (29.2)	143 (73.3)
Histological subtype				
NST	64 (32.8)	39 (20.0)	75 (38.5)	178 (91.3)
Other	13 (6.7)	1 (0.5)	3 (1.5)	17 (8.7)
Grade				
1	16 (8.2)	3 (1.5)	3 (1.5)	22 (11.3)
2	53 (27.2)	29 (14.9)	50 (25,6)	132 (67.7)
3	8 (4.1)	8 (4.1)	25 (12.8)	41 (21.0)
Stage				
IIA	8 (4.1)	4 (2.1)	4 (2.1)	16 (8.2)
IIB	17 (8.7)	6 (3.1)	20 (10.3)	43 (22.1)
IIIA	25 (12.8)	11 (5.6)	17 (8.7)	53 (27.2)
IIIB	27 (13.8)	19 (9.7)	37 (19.0)	83 (42.6
Chemotherapy regimen				
Anthracycline/taxane	72 (36.9)	26 (13.3)	70 (35.9)	168 (86.2)
Anthracycline/taxane + trastuzumab	-	13 (6.7)	-	13 (6.7)
Anthracycline/taxane + carboplatin	-	-	3	3 (1.5)
Anthracycline + cyclophosphamide	6 (3.1)	1 (0.5)	4 (2.0)	11 (5.6)
Response				
pCR	18 (9.2)	17 (8.7)	21 (10.8)	56 (28.7)
No pCR	60 (30.8)	22 (11.3)	57 (29.2)	139 (71.3)

NST, no special type; pCR, pathological complete response.

P<0.05 was considered to indicate a statistically significant difference.

Results

Clinicopathological characteristics. A total of 195 patients met the selection criteria and were included in the study. The mean age was 46.3±10.4 years and 26.7% of patients were aged <40 years. Invasive carcinoma of no special type was the predominant histology (91.3%). Most tumors were grade 2 (67.7%) and stage III (69.8%). Immunohistochemistry revealed that 39.5% of the tumors were positive for ER and/or PR, 8.7% were positive for both HER2 and ER and/or ER, 11.8% were positive for HER2 only and 40.0% were TN (data not shown). The molecular subtypes were luminal (39.5%), HER2-positive (20.5%) and TN (40%). NAC was primarily based on sequential anthracycline-taxane regimens (94.4%). The number of cycles ranged from six to eight in 91.3% of patients (data not shown). Among HER2-positive patients, 32.5% received anti-HER2 therapy (trastuzumab) as part of the NAC regimen. In the remaining HER2-positive patients (67.5%), trastuzumab was given in the adjuvant setting (following surgery). Among TN patients, a platinum agent (carboplatin) was added to the NAC regimen in 3.8% of cases. Mastectomy was performed in 75.4% of patients and 95.9% received postoperative radiotherapy. Endocrine therapy was administered to all patients with hormone receptor-positive tumors, while adjuvant capecitabine was given to patients with TN tumors without pCR (data not shown). pCR was observed in 28.7% of patients based on pathological examination of the surgical specimen (Table I).

Association between pCR and clinicopathological variables. Molecular subtype and tumor stage influenced the pathological response. Patients with HER2-positive tumors were more likely to achieve pCR than those with TN subtypes [odds ratio (OR), 2.62; 95% CI, 1.10-6.22). Patients with stage II tumors were also more likely to achieve pCR than those with stage III tumors (OR, 3.67; 95% CI, 1.82-7.48). However, age, grade and pathological subtype did not significantly influence the pathological response (Table II).

Univariate analysis. The 5-year PFS and OS rates were 68.4% (median, 40.23 months) and 78% (median, 42.13 month), respectively, with a median follow-up of 49.4 months. Stage and pathological response significantly influenced the 5-year PFS and OS rates. Patients with stage II tumors had significantly higher 5-year PFS and OS rates than patients with stage III tumors (81.9 vs. 62.7%; and 93 vs. 71.2%, respectively). pCR was associated with significantly higher 5-year PFS and OS rates than no pCR (96.1 vs. 57.7%; and 95 vs. 71.6%, respectively; Table III).

Table II. Logistic regression analysis of the association between pathological response and clinicopathological variables.

Variable	No pCR, n (%)	pCR, n (%)	P-value	OR	95% CI
Age, years			0.231		
<40	33 (16.9)	19 (9.7)		1.57	0.75-3.33
≥40	106 (54.4)	37 (19.0)		1	-
Pathological subtype			0.546		
NST	125 (64.1)	53 (27.2)		1.53	0.38-6.17
Other	14 (7.2)	3 (1.5)		1	-
Stage			<0.001		
II	32 (16.4)	27 (13.8)		3.67	1.82-7.48
III	107 (54.9)	29 (14.9)		1	-
Grade			0.382		
1	19 (9.7)	3 (1.5)		0.35	0.08-1.56
2	93 (47.7)	39 (20.0)		0.71	0.31-1.64
3	27 (13.8)	14 (7.2)		1	-
Molecular subtype			0.048		
Luminal	60 (30.8)	17 (8.7)		0.94	0.41-2.11
HER2-positive	22 (11.3)	18 (9.2)		2.62	1.10-6.22
Triple-negative	57 (29.2)	21 (10.8)		1	-

pCR, pathological complete response; OR, odds ratio; NST, no special type.

Table III. Univariate analysis by the log-rank test.

Variable	5-year PFS (%)	P-value	5-year OS (%)	P-value
Age, years		0.452		0.562
≤40	69.5		84.6	
>40	67.7		75.5	
Histological subtype		0.623		0.787
NST	70.4		77.1	
Other	53.3		86.7	
Grade		0.771		0.396
1	55.1		84.8	
2	69.8		78.9	
3	71.5		69.9	
Molecular subtype		0.389		0.371
Luminal	70.7		83.1	
HER2-positive	68.9		79.9	
Triple-negative	64.7		71.9	
Stage		0.005		0.006
II	81.9		93	
III	62.7		71.2	
Response		<0.001		0.002
pCR	96.1		95	
No pCR	57.7		71.6	

PFS, progression-free survival; OS, overall survival; NST, no special type; pCR, pathological complete response.

Multivariate analysis. Tumor stage significantly influenced the 5-year OS but not the 5-year PFS [hazard ratio (HR), 0.32; 95% CI, 0.11-0.93 for OS; and HR, 0.47; 95% CI, 0.22-1.02 for PFS. Pathological response significantly influenced both

Table IV. Multivariate analysis by the Cox model.

	Progression-free survival			Overall survival		
Variable	P-value	HR	95% CI	P-value	HR	95% CI
Stage						
II	0.055	0.47	0.22-1.02	0.036	0.32	0.11-0.93
III		1.00	-		1	-
Response						
pCR	0.002	6.54	2.02-21.19	0.016	5.84	1.39-24.54
No pCR		1	-		1	-

HR, hazard ratio; pCR, pathological complete response.

5-year PFS and OS (HR, 6.54; 95% CI, 2.02-21.19 for PFS; HR, 5.84; 95% CI, 1.39-24.54 for OS; Table IV).

Survival rate according to pathological response in patients with different molecular subtypes. Within each molecular subtype group, the 5-year PFS rate of the pCR group was significantly higher than that of the no pCR group [100.0 vs. 64.2% for the luminal subtype; 94.4 vs. 49% for the HER2-positive subtype; and 94.4 vs. 53.5% for the TN subtype; Fig. 1).

The pCR group had a significantly higher 5-year OS rate than the no pCR group in patients with the TN subtype (64.3 vs. 94.4%;). In patients with the luminal and HER2-positive subtypes, the 5-year OS rate of the pCR group was higher than that of the no pCR group, but the difference was not significant (100.0 vs. 78.9% for luminal subtype; and 91.7 vs. 70.9% for HER2-positive subtype, respectively; Fig. 2).

Discussion

The aim of the present study was to evaluate the response to NAC and determine its impact on survival rates in patients with breast cancer. Approximately one-third of patients experienced pCR following NAC. This result is similar to that of Farrukh *et al* (18), who reported a pCR rate of 27.2%, using the same definition as the present study (absence of residual invasive or micro-invasive disease in both the breast and axillary lymph nodes, residual in situ disease). Cirier *et al* (19) and Cortazar *et al* (20) obtained lower pCR rates of 16 and 13%, respectively. The definition of pCR in the aforementioned studies (the absence of any invasive or *in situ* carcinoma) may explain the low pCR rates. However, Müller *et al* (21), using the same definition as the present study, reported a higher pCR rate (47%) than the present study. The chemotherapy regimen and drugs (anti-HER2) used in the aforementioned study could explain the high pCR rate. Studies have shown that the use of anti-HER2 therapy increases the pCR in HER2-positive cancer (22-24). In TN breast cancer, combination of platinum agents with conventional chemotherapeutic agents (anthracyclines and taxanes) has also been shown to be superior to sole conventional therapeutic agents (25). Adding carboplatin to anthracycline/taxane regimens improves pCR in early-stage TN breast cancer (25). In the present study, one-third of HER2-positive patients received anti-HER2 therapy as part of

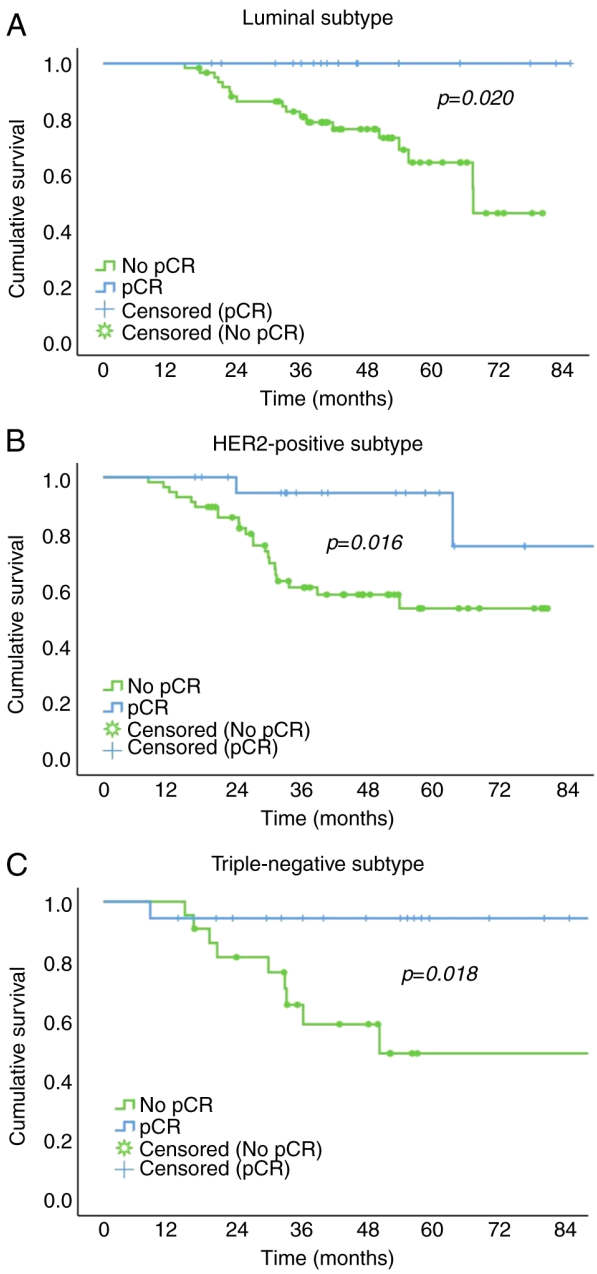

Figure 1. Progression-free survival curves according to pathological response in patients with different molecular subtypes. (A) Luminal. (B) HER2-positive. (C) Triple-negative. pCR, pathological complete response.

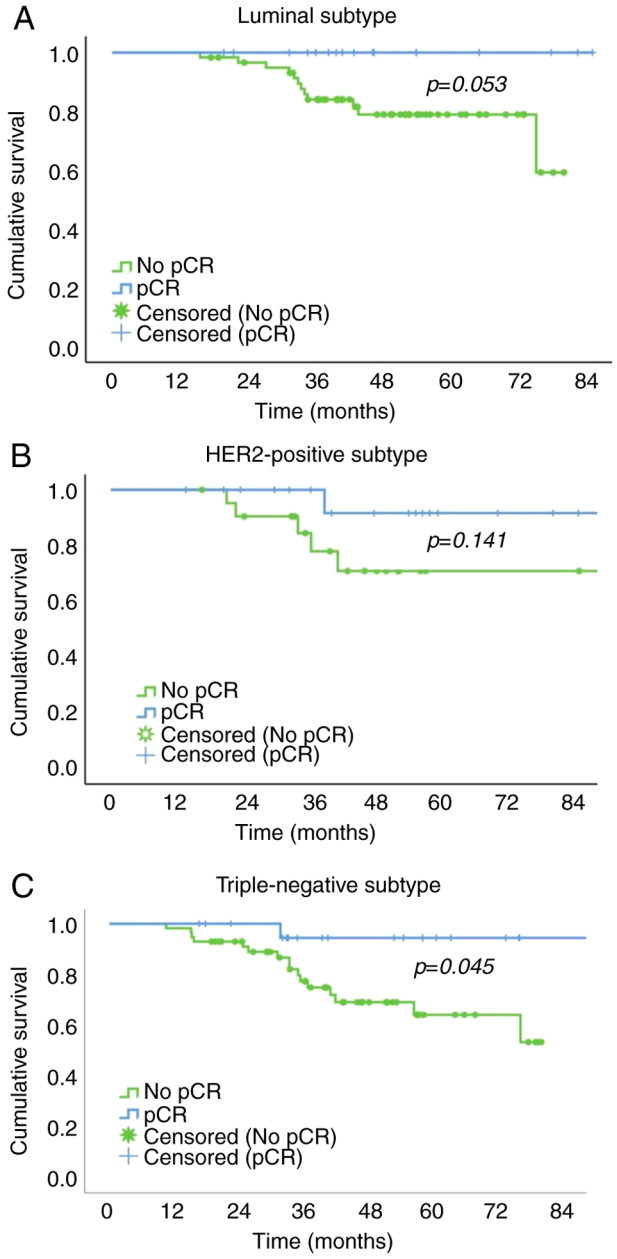

Figure 2. Overall survival curves according to pathological response in patients with different molecular subtypes. (A) Luminal. (B) HER2-positive. (C) Triple-negative. pCR, pathological complete response.

their chemotherapy regimen, and a small percentage (1.5%) of patients with TN breast cancer received carboplatin. This was because immunohistochemistry results of most patients were not available at the time of NAC.

In addition to the definition of pCR and the chemotherapy regimen, other clinical and pathological factors influence response rates. Several studies have identified tumor biology as the strongest factor influencing the probability of achieving pCR (6,26-29). HER2-positive and TN breast cancers are more likely to respond to NAC compared to hormone receptor-positive cancers (6,29). In the present study, HER2-positive tumors were associated with a higher probability of pCR compared to triple negative . The present results were similar to those of Swain *et al* (7). TN breast cancer was an important concern in the present study owing to its high frequency. In contrast to occidental and east and south African series (12,30,31), the present study reported a high rate of TN breast cancer (40%) similar to other west African studies (32,33). probability of pCR in TN tumors was not significantly different from luminal subtype. This may be explained by the molecular heterogeneity of TN breast cancer. According to Lehmann *et al* (34), TN breast cancers can be subdivided into four subtypes [basal-like 1 and 2, mesenchymal and luminal androgen receptor (AR)], which have different clinical and pathological characteristics. The basal-like 1 group demonstrates the highest response rate to NAC, while the basal-like 2 group demonstrates the lowest response rate. Most studies that have demonstrated a high rate of pCR in patients with TN breast cancer mostly included non-Black or non-African patients (6,10,35). The present sample was composed of Black African patients, whose molecular characteristics differ from those of White or Asian patients (10,36). However, a recent study by Rajagopal *et al* (13) found that young Black female patients have similar proportions of basal-like 1 and mesenchymal subtypes to European or Asian patients, a smaller relative proportion of luminal AR-type tumors and a larger proportion of non-subtyped tumors. Independent of the Lehmann classification of triple-negative breast cancers (TNBC), a distinct subgroup lacking androgen receptor (AR) expression referred to as quadruple-negative breast cancer (QNBC) has been identified (37,38). This subtype is predominantly observed in West African and African-American populations. (36). Notably, the prognostic impact of AR negativity appears to vary between ethnic and racial groups (39). In West African (particularly Nigerian), African-American, and Asian women, the absence of AR expression has been associated with chemoresistance and reduced overall survival (39-41).

In the present study, tumor stage influenced the pathological response. Stage II was associated with a significantly higher probability of pCR than stage III. The present results are consistent with those of Goorts *et al* (42), who demonstrated that tumor stage was an independent and strong predictor of pCR compared with HER2, ER and PR status, and grade.

In the present study, pCR was the sole independent predictive factor for improved PFS and OS. The molecular subtype did not influence OS or PFS. However, achieving a pCR was associated with a significantly improved 5-year PFS rate in each molecular subtype. In the TN group, pCR was associated with significantly improved 5-year OS. The present findings are consistent with those of Cortazar *et al* (20), who found an increase in event-free survival with pCR in each molecular subtype, although this was weakest for hormone receptor-positive and low-grade tumors. Spring *et al* (43) found that pCR was associated with improved OS and event-free survival in the TN and HER2-positive subtypes. von Minckwitz *et al* (5) demonstrated that pCR improved disease-free survival in luminal B/HER2-negative, HER2-positive/non-luminal and TN tumors, but not in luminal or luminal B/HER2-positive cancer. When evaluating the long-term results of the Alliance study (trial no. ACOZOG Z1071), Boughey *et al* (26) found that breast cancer-specific survival rates were higher in the pCR group compared with no pCR (residual disease) for the hormone receptor-positive and TN groups, but not for the HER2-positive group.

To the best of our knowledge, the present study was the first to evaluate the impact of pathological response to NAC on breast cancer survival in patients from the Republic of Côte d'Ivoire. However, it has limitations that should be noted, including the retrospective nature of the study, the absence of centralized pathological exams and the absence of Ki67 testing. The pathological exams were performed in different laboratories with different techniques and preparation conditions. A centralized examination would allow the specimens to be subjected to the same preparation conditions to ensure the robustness of the results. The retrospective nature of the present study prevented control of specimen handling and NAC administration. The proliferation index Ki67 data, which distinguishes between low- and high-grade cancer, were not available. In the Republic of Côte d'Ivoire, immunohistochemistry testing of endocrine (estrogen and progesterone) receptors and HER2, but not Ki67, is free for patients with breast cancer. Therefore, Ki67 testing was not systematically performed. Consequently, the intrinsic subtype classification did not differentiate cancers into luminal A and B, nor into HER2-positive/hormone receptor-positive and HER2-positive/hormone receptor-negative.

In the present study, pCR after NAC in breast cancer was found to be a positive predictor of progression-free and OS. While previous studies found a higher pCR rate in the TN subgroup (6,29), the present study found that the probability to achieve pCR was not significantly different between TN and luminal subgroups. This indicated that TN breast cancer was less sensitive to chemotherapy, potentially due to predominant expression of AR in TN tumors. Future studies should investigate the genetics and phenotype of TN cancer.

Although the TN subtype was not significantly associated with a higher probability of pCR rate than the other subgroups, reaching a pCR resulted in significantly improved OS. These findings support the use of intensify NAC regimens for this molecular subgroup to improve survival rates. Immune checkpoint inhibitors have proven efficacy with an acceptable toxicity profile in neoadjuvant treatment of TN tumors (44). However, these chemotherapeutic agents are not currently available in the Republic of Côte d'Ivoire. Access to immune checkpoint inhibitors may improve survival rates in patients with breast cancer. Pertuzmab in combination with chemotherapy resulted in a higher response rate in HER2-positive breast cancer (23). In the Republic of Côte d'Ivoire, anti-HER2 therapies are available free of charge (since 2018 for trastuzumab and 2024 for pertuzumab). However, delays in immunohistochemistry testing due to lack of facilities and qualified personnel hamper the use of anti-HER2 therapy and platinum agents in HER2-positive and TN patients, respectively. Centralization of pathology services and personal training programs may address this problem.

In conclusion, NAC for breast cancer resulted in pCR in approximately one-third of patients. Tumors positive for HER2 demonstrated the highest probability of pCR following neoadjuvant chemotherapy. A pathological response following NAC was the most relevant factor influencing PFS and OS in all patients. For each molecular subtype, pCR was associated with a significantly higher PFS rate. Therefore, the pathological response following NAC was the strongest predictive factor of improved PFS, particularly in the TN subtype.

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Availability of data and materials

The data generated in the present study may be requested from the corresponding author.

Authors' contributions

ENS designed the study, analyzed and interpreted data, and wrote the manuscript. DAT designed the study and wrote the manuscript. CTS, AGT and KT analyzed and interpreted data. BD conceived the study. AGT and DAT confirmed the authenticity of all the raw data. All authors have read and approved the final version of the manuscript.

Ethics approval and consent to participate

The present study was approved by The National Ethical Committee of Life and Health Sciences (approval no. 00068/25/MSHPCMU/CNESVS-km; Côte d'Ivoire. All patients gave their written consent to participate to the study.

Patient consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Use of artificial intelligence tools

During the preparation of this work, artificial intelligence tools were used to improve the readability and language of the manuscript, and subsequently, the authors revised and edited the content produced by the artificial intelligence tools as necessary, taking full responsibility for the ultimate content of the present manuscript.

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