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Retrospective Evaluation of Metabolic Syndrome's Role in Breast Cancer Outcomes in Black Women

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ABSTRACT

Background: Metabolic Syndrome (MS) is a cluster of chronic conditions characterized by the co-occurrence of at least three out of five clinical risk factors: central obesity, hyperglycemia, hypertension, elevated circulating triglycerides, and low high-density lipoprotein (HDL) levels. MS is a well-established precursor to cardiovascular disease and type 2 diabetes mellitus (DM), but emerging evidence also links MS-related pathways, particularly those involving obesity and hyperglycemia, to increased breast cancer (BC) risk and tumor progression. Despite this growing body of literature, there remains a significant gap in understanding the interplay between MS components and BC progression among Black women, a population disproportionately affected by both MS and adverse breast cancer outcomes. **Objective:** This study aims to evaluate the relationship between DM and BC among Black women in an ambulatory care setting, with a specific focus on the timing of diabetes onset in relation to BC diagnosis and treatment. **Methods:** We conducted a retrospective, longitudinal cohort study using medical records from an outpatient oncology clinic in an urban university hospital. The study population included self-identified Black women who were diagnosed with BC between the ages of 18 and above and received care at our institution over a three-year period. Data extracted included age at BC diagnosis, diabetes status, body mass index (BMI), timing of diabetes onset, and treatment history, including chemotherapy and hormonal therapy. **Results:** Among the study cohort, the median age of BC diagnosis in Black women was 60 years, which is notably younger than the average age of BC diagnosis in the general global population. Only 24% of the Black women with BC had a prior diagnosis of diabetes at the time of cancer diagnosis, while the remaining 76% were non-diabetic. Interestingly, 71% of the women who had both BC and DM developed diabetes only after the diagnosis of BC and initiation of hormonal therapy, suggesting a potential iatrogenic or disease-related metabolic shift. Additionally, 71% of the total BC patients in the cohort were overweight (BMI ≥ 25 kg/m²), and 34% of them were categorized as obese (BMI ≥ 30 kg/m²), further supporting the hypothesis that excess weight plays a pivotal role in the development of both BC and DM. **Conclusions:** Our findings suggest that while a minority of Black women with BC had pre-existing diabetes, the majority developed DM after their diagnosis of BC and initiation of hormonal therapy, indicating a possible treatment-related or disease-associated onset. Furthermore, the high prevalence of overweight and obesity in the cohort underscores the role of excess adiposity as a central driver in the development of breast cancer and subsequent metabolic disturbances. These results emphasize the

need for proactive metabolic screening and weight management interventions in Black women diagnosed with BC, particularly during and after cancer treatment, to mitigate the risk of DM and improve long-term health outcomes.

Keywords: *Metabolic Syndrome, Breast cancer, Diabetes Mellitus, Black Women, Obesity*

INTRODUCTION

Breast cancer (BC) remains a major public health concern in the United States. According to the American Cancer Society's 2024 report, BC is the most commonly diagnosed cancer among women and the second leading cause of cancer-related death, surpassed only by lung cancer[1]. In 2024 alone, approximately 310,720 new cases of BC are expected to be diagnosed, and an estimated 42,250 women are projected to die from the disease[2]. Despite the overall incidence of BC being slightly lower in Black women (127.1 per 100,000) compared to white women (132.5 per 100,000), the mortality rate tells a different and alarming story. Black women experience a 41% higher BC-related mortality compared to their white counterparts[2,3]. This disparity is not fully explained by differences in access to care or socioeconomic status alone and suggests a complex interplay of biological, metabolic, and treatment-related factors.

One contributing factor to the increasing incidence of breast cancer, particularly among postmenopausal women, is the rising prevalence of obesity, coupled with lifestyle shifts such as decreased fertility and physical inactivity[4-6]. Obesity is not only a risk factor for BC development, but it also influences disease progression and treatment outcomes, especially in metabolically vulnerable populations.

Metabolic syndrome (MS) is a cluster of interrelated metabolic disturbances, including insulin resistance (IR), central obesity, hyperglycemia, hypertension, and dyslipidemia[7,8]. It significantly increases the risk for cardiovascular disease and type 2 diabetes mellitus (DM), and growing evidence implicates MS in the etiology and progression of BC[9,10]. Specifically, MS may promote BC through dysregulated hormonal pathways involving insulin, estrogen, inflammatory cytokines, and growth factors. In the setting of IR or type 2 DM, elevated insulin levels and impaired glucose metabolism promote fat accumulation. This excess adiposity further worsens insulin sensitivity, creating a vicious cycle that exacerbates both metabolic dysfunction and oncogenic signaling[11].

Notably, the relationship between obesity and BC is paradoxical and influenced by menopausal status. Obesity is generally considered protective in premenopausal women but increases BC risk in postmenopausal women[12]. In postmenopausal women, adipose tissue becomes the primary site for estrogen production via aromatization of androgens. Elevated estrogen levels bind to estrogen receptors in breast epithelial cells, stimulating uncontrolled proliferation and increasing the risk of hormone receptor-positive breast cancer[13,14]. Furthermore, menopause itself is associated with weight gain, further compounding this risk[15].

Hormonal therapy is a cornerstone of treatment for hormone receptor-positive BC. Tamoxifen is used in both pre- and postmenopausal women, whereas aromatase inhibitors (AIs) such as anastrozole, letrozole, and exemestane are primarily used in postmenopausal patients[16]. AIs function by inhibiting aromatase, the key enzyme responsible for estrogen biosynthesis in peripheral tissues[17]. However, emerging studies now suggest that these therapies, while effective in reducing BC recurrence, may significantly increase the risk of developing diabetes[18]. One study reported a 2.2-fold increase in diabetes risk among tamoxifen users and a 4.3-fold increase among those on aromatase inhibitors, compared to non-users[19].

Given the high prevalence of metabolic syndrome and obesity in Black women and their disproportionate burden of aggressive BC and poorer outcomes, investigating the metabolic impact of BC and its treatment in this population is urgently needed. This study addresses that gap by evaluating the relationship between diabetes mellitus and breast cancer in Black women, with attention to timing, treatment, and metabolic risk factors.

Objective

DM is present in approximately one-third of patients with BC and is independently associated with increased cancer-related mortality[20]. Simultaneously, obesity - a key driver of type 2 DM and a modifiable risk factor for both BC incidence and progression - disproportionately affects Black women compared to other racial and ethnic groups in the United States[21]. Despite having a slightly lower incidence of BC, Black women experience significantly higher mortality rates than white women, suggesting the presence of underlying biological and systemic factors that exacerbate outcomes.

Given this intersection of metabolic risk, racial disparity, and poor prognosis, our study aimed to investigate the association between DM and BC specifically in Black women. We examined the timing and prevalence of diabetes relative to BC diagnosis, its potential role in the progression of disease, and its contribution to adverse outcomes. By focusing on this understudied and underserved population, we sought to better understand how metabolic dysfunction,

particularly diabetes, may influence the burden and trajectory of breast cancer in Black women and to identify potential opportunities for earlier intervention and improved survivorship.

MATERIAL AND METHODS

Study Design and Population

This retrospective longitudinal cohort study was conducted using electronic medical records (EMRs) from the outpatient Diabetes Treatment Center and Oncology Clinic at a major urban medical center. All data were de-identified before analysis to ensure patient confidentiality. The study population included women aged 18 years and older with a confirmed diagnosis of BC who received care between October 2019 and September 2022.

Exclusion criteria included patients younger than 18 years of age, individuals without a confirmed BC diagnosis, and those with insufficient follow-up, defined as fewer than 80% of scheduled visits in either the Diabetes or Oncology clinics during the study period. The study protocol was reviewed and approved by the University's Institutional Review Board (IRB) on October 10, 2022. The study protocol was reviewed and approved by the University's IRB under protocol number **[IRB-2022-0672]**. All patient data were de-identified prior to analysis and securely stored in compliance with institutional data protection policies and HIPAA regulations. Given the retrospective nature of the study and use of de-identified data, the IRB granted a waiver of informed consent.

Data Sources and Collection

Patient data were extracted from EMRs using two platforms: Clinipro (in the Diabetes Treatment Center) and Allscripts (in the Oncology Ambulatory Clinics). We identified eligible patients with BC and/or DM who visited the outpatient clinics during the study timeframe (October 2019 to September 2022). Key demographic and clinical information were systematically reviewed and recorded.

The BC diagnosis index date was defined as the first documented diagnosis in the EMR (pathology, oncology notes, or problem list). Endocrine therapy initiation was approximated using the calendar year of BC diagnosis and evidence of aromatase inhibitor or tamoxifen use. Patients were followed from diagnosis until the earliest of last clinic visit, death, or administrative censoring (September 30, 2022). Median follow-up was 108 months (IQR: 84–144). *Diabetes diagnosis ascertainment was defined as follows: prevalent DM was* diagnosed within 24 months prior to BC diagnosis, i*ncident DM prior to therapy was d*iagnosed between BC diagnosis and therapy initiation, and i*ncident DM during therapy was d*iagnosed from therapy start through follow-up. A sensitivity analysis evaluated DM incidence within the first 180 days of therapy. Progression of breast cancer was defined as worsening AJCC stage or documentation of recurrence/metastasis. Events within 30 days of diagnosis were excluded to avoid misclassification. The progression window extended from day 31 post-diagnosis through censoring.

Study Outcomes and Definitions

The primary independent variables were DM status (prevalent vs. incident), Timing of DM onset (before, concurrent with, or after breast cancer diagnosis), Exposure to endocrine therapy (type and timing). The primary dependent variables included age, body mass index (BMI), glycemic status, breast cancer treatment regimen, and BC progression, which was defined as a change in cancer stage from initial diagnosis to follow-up.

Patients were categorized as overweight if BMI was >25 kg/m² and obese if BMI was ≥30 kg/m². Menopausal status was stratified using an age cutoff: women younger than 51 years were classified as premenopausal, and those 51 years or older as postmenopausal. Additional baseline covariates included BC histologic subtype, year of diagnosis, comorbid conditions, prescribed medications, and race/ethnicity.

Primary outcomes assessed were whether there was progression of breast cancer, the time to progression where applicable, and time to incident diabetes mellitus.

Statistical Analysis

Descriptive statistical methods were used to analyze the data. Baseline characteristics of the study cohort were summarized using measures of central tendency and frequency distributions. We conducted preliminary comparative analyses to evaluate the relationship between diabetes mellitus (DM), metabolic risk factors, and BC progression among African American women. Mean differences in age, BMI, and disease progression metrics were evaluated to explore potential associations between metabolic status and cancer outcomes.

A multivariable logistic regression model was used to assess the association between diabetes status and breast cancer progression. Predictors included diabetes status and age at diagnosis, body mass index, pre- or post- menopausal, cancer stage at diagnosis, and type of cancer treatment. Effect sizes were reported as odds ratios (ORs) with 95% confidence intervals (CIs). Although not implemented in this pilot phase, Cox proportional hazards models are planned for future analyses to evaluate time-to-progression and time-to-incident DM. Duration will be calculated as the difference between the year of diagnosis and the end of the study period (2022), with hazard ratios (HRs) and 95% CIs reported.

Covariates were prespecified based on clinical relevance. Cases with missing values in key variables were

excluded from the regression model. For future analysis, multiple imputations will be applied to address missing data in BMI, therapy type, and staging variables.

A post hoc power analysis was conducted to assess sample adequacy. Based on observed progression rates (27% in diabetic vs. 8% in non-diabetic patients) and a calculated effect size (Cohen's h = –0.796), the achieved statistical power was 0.189, substantially below the conventional threshold of 0.80. This indicates the study was underpowered to detect statistically significant differences, largely due to the small sample size, particularly in the non-diabetic subgroup. Fisher's exact test was used to compare breast cancer progression rates between diabetic and non-diabetic Black women, given the small sample size and binary outcome distribution.

RESULTS

Patient Baseline Demographic and Clinical Characteristics

A total of 68 patient medical records were reviewed. Among them, 16 patients (23.5%) had a documented diagnosis of DM, while 52 patients (76.5%) did not. The median age at BC diagnosis was 53 years in the diabetes group and 60 years in the non-diabetes group.

Given the average age of menopause in the U.S. is 51 ± 12.6 years, patients were stratified by menopausal status using age 51 as the cutoff. Based on this definition, 46 patients (67.6%) were considered postmenopausal at the time of BC diagnosis.

Regarding BMI, 48 patients (70.6%) had a BMI >25 kg/m², indicating overweight or obesity, with 26 (38.2%) of these classified as obese (BMI ≥30 kg/m²). BMI data was unavailable for two participants.

The baseline demographic and clinical characteristics of all study participants are summarized in **Table 1**.

Patient Baseline Characteristics of Black Participants

Of the total study population, 58 participants (85.3%) identified as Black and had a diagnosis of breast cancer. Among these, 14 individuals (24%) had a documented diagnosis of diabetes mellitus, while 44 (76%) did not.

Based on the menopausal age cutoff of 51 years, 38 Black participants (65.5%) were classified as postmenopausal at the time of BC diagnosis. In terms of BMI, 41 participants (70.6%) had a BMI greater than 25 kg/m², indicating overweight or obesity, and 20 participants (34%) met the criteria for obesity (BMI ≥30 kg/m²).

These baseline demographic and clinical characteristics for Black participants are summarized in **Table 1**.

Breast Cancer Progression in Diabetic and Non-Diabetic Participants

To evaluate breast cancer progression in relation to diabetes status, we compared outcomes among Black women with and without diabetes. A total of 23 participants experienced breast cancer progression during the study period, as summarized in Table 1. Of these, 11 individuals (47.8%) had a diagnosis of DM. At baseline, the majority of patients (13 participants, 56.5%) were diagnosed with Stage I breast cancer, while only one participant (4.3%) was diagnosed at Stage IV.

As illustrated in **Figure 1**, disease progression was more pronounced among participants with diabetes (n=11). Specifically, 27% of women in the BC with DM group experienced progression of disease (n=3), compared to only 8% (n=1) of the 12 in the non-diabetic group. Conversely, 92% of participants in the non-diabetes group showed no evidence of cancer progression, in contrast to 73% in the diabetes group who remained stable. These findings suggest that the presence of diabetes may be associated with a higher likelihood of breast cancer progression, as assessed by changes in cancer staging over time.

Endocrine Therapy and Cancer Chemotherapy and Secondary Diabetes in Black Women

Among the Black women with BC included in this study, 14 participants (24%) had a documented comorbidity of DM, as shown in **Table 1**. Notably, 71% of these women were diagnosed with BC before developing DM, while 21% had a pre-existing diagnosis of DM. One patient was diagnosed with both conditions concurrently.

The majority of Black participants with BC and DM (10 patients, 71.4%) were prescribed aromatase inhibitors, primarily anastrozole or letrozole, as part of their cancer treatment regimen. Among those receiving aromatase inhibitors, approximately 70% developed secondary diabetes following the initiation of therapy.

The median follow-up time was 108.0 months (IQR: 60.0 months). Fisher's exact test yielded a p-value of 0.29 (**Table 2**), indicating no statistically significant difference in progression rates between groups. Attempts to perform adjusted logistic regression were unsuccessful due to perfect separation and singular matrix errors, likely driven by the small sample size and sparse progression events. These limitations are noted in the Discussion, and we emphasize the need for larger, adequately powered studies to validate these findings and further explore the potential metabolic impact of endocrine therapy. These findings suggest a potential association between aromatase inhibitor therapy and the onset of secondary diabetes in Black women with BC. A detailed summary of participant treatment and diabetes onset is provided in **Table 3**.

The overall retrospective study design and patient flow are illustrated in **Figure 2**

Table 1. Baseline Demographic and Clinical Characteristics of Participants

Characteristics	Diabetes (N, %)	Nondiabetic (N, %)	Total (N, %)
Total number	16 (23.5)	52 (76.5)	68 (100)
Age at the time of diagnosis of BC (Mean ± SD)	56.9 ± 16.4	55.8 ± 11	
Age of diagnosis of BC (Median)	53	60	
< 51 years	4 (5.88)	17(25)	21 (30.88)
≥ 51 years	12 (17.64)	34 (50)	46 (67.64)
BMI			
< 25 kg/m^2	2 (2.94)	16 (23.52)	18 (26.42)
25 – 30 kg/m^2	4 (5.88)	18 (26.47)	22 (32.35)
> 30 kg/m^2 (obese)	10 (14.7)	16 (23.52)	26 (38.22)
25 kg/m^2 and above	14 (20.58)	34 (50)	48 (70.58)
Unknown		2 (2.94)	2 (2.94)
Black patients			
Total number	14 (24)	44 (76)	58 (100)
Age at the time of diagnosis of BC (Mean ± SD)	56 ±17.3	56 ± 11.2	
Age of diagnosis of BC (Median)	53	60	
< 51 years	4 (6.89)	15 (25.86)	19 (32.75)
≥ 51 years	10 (17.24)	28 (48.27)	38 (65.51)
BMI			
< 25 kg/m^2	2 (3.44)	14 (24.13)	16 (27.57)
25 – 30 kg/m^2	4 (6.88)	17 (29.3)	21 (36.18)
> 30 kg/m^2	8 (13.76)	12 (20.68)	20 (34.44)
25 kg/m^2 and above	12 (20.68)	29 (50)	41 (70.68)
Black patients with data on BC progression			
Total number	11 (47.8)	12 (52.2)	
Age at the time of diagnosis of BC (Mean ± SD)	58.5 ± 18.2	56.16 ± 11.6	
Median age of diagnosis of BC (Median)	53	60	
> 51 years	2 (8.69)	3 (13)	5 (21.69)
≥ 51 years	9 (39.13)	9 (39.13)	18 (78.26)
BMI			
< 25 kg/m^2			
25 – 30 kg/m^2	2 (8.69)	3 (13.04)	5 (11.73)
> 30 kg/m^2	3 (13.04)	0 (0)	3 (13.04)
25 kg/m^2 and above	6 (26.08)	9 (39.13)	15 (65.21)
	9 (39.13)	9 (39.13)	18 (78.6)
Cancer stage at diagnosis			
I	8 (34.78)	5 (21.7)	13 (56.48)
II	1 (4.34)	5 (21.7)	6 (26.04)
III	2 (8.68)	1 (4.34)	3 (13.02)
IV	0 (0)	1 (4.34)	1 (4.34)

***BMI**: Body Mass Index, **SD**: Standard Deviation, **BC**: Breast Cancer, **DM**: Diabetes Mellitus*

Table 2. Fisher's Exact Test of Breast Cancer Progression in Diabetic vs. Non-Diabetic Black Women

Group	Progression	No Progression	Total
Diabetic Black women	3	7	10
Non- Diabetic Black women	1	11	12
Total	4	18	22

Two tailed Fisher's exact p-value P=0.29
Median follow up = 108 months (IQR 84-144)

IQR – Interquartile range

Table 3. Endocrine Therapy and Cancer Chemotherapy: Secondary Diabetes in Black Women

Clinical Characteristics	Total Number of patients (N, %)	Total number of Black patients (N, %)	Total number of Black patients with data on BC progression (N, %)
BC + DM	16 (100)	14 (100)	11 (100)
BC before DM	12 (75)	10 (71.4)	8 (72.7)
BC after DM	3 (18.75)	3 (21.4)	2 (18.18)
BC same time as DM	1 (6.25)	1 (7.14)	1 (9.09)
Endocrine Therapy and Cancer Chemotherapy			
i. Aromatase Inhibitors	**12**	**10**	**8**
Anastrozole	10 (83.3)	9 (90)	7 (87.5)
Letrozole	2 (16.66)	1 (10)	1 (12.5)
ii. Antimetabolites			
Capecitabine	1	1	1
iii. None	3	3	2
DM after Endocrine Therapy and Cancer Chemotherapy (Secondary Diabetes)			
i. Aromatase inhibitors	**9/12 (75)**	**7/10 (70)**	**6/8 (75)**
Anastrozole	7/10 (70)	6/9 (66.66)	5/7 (71.4)
Letrozole	2/2 (100)	1/1 (100)	1/1 (100)
ii. Antimetabolite			
Capecitabine	0	0	0
iii. None	3	3	2

BC – Breast Cancer, DM – Diabetes Mellitus, N, % - Number of patients (percentage of patients)

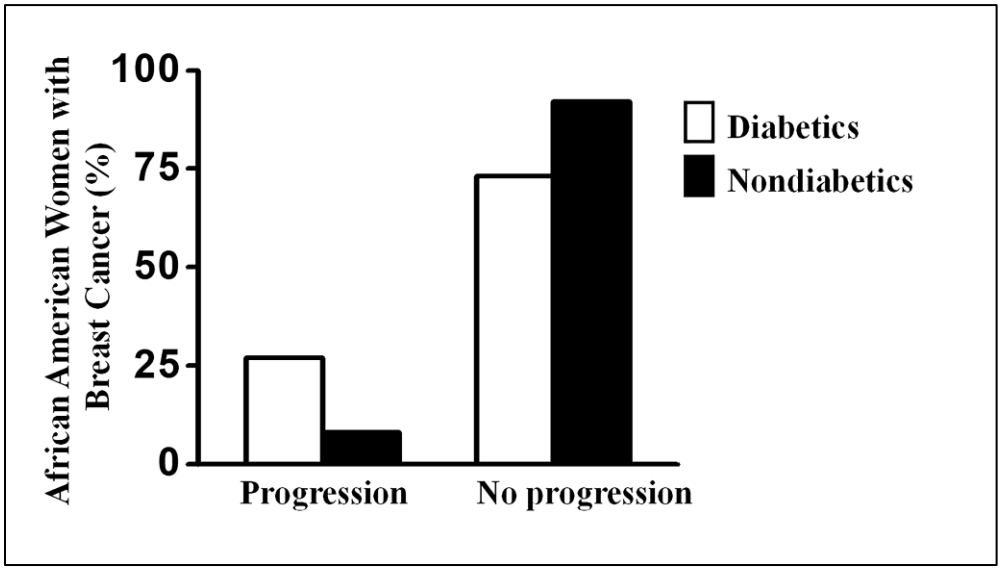

Figure 1. Comparison of Breast cancer progression between diabetic and non-diabetic Black women. The empty bar indicates diabetics. The black bar indicates nondiabetic Black women with breast cancer.

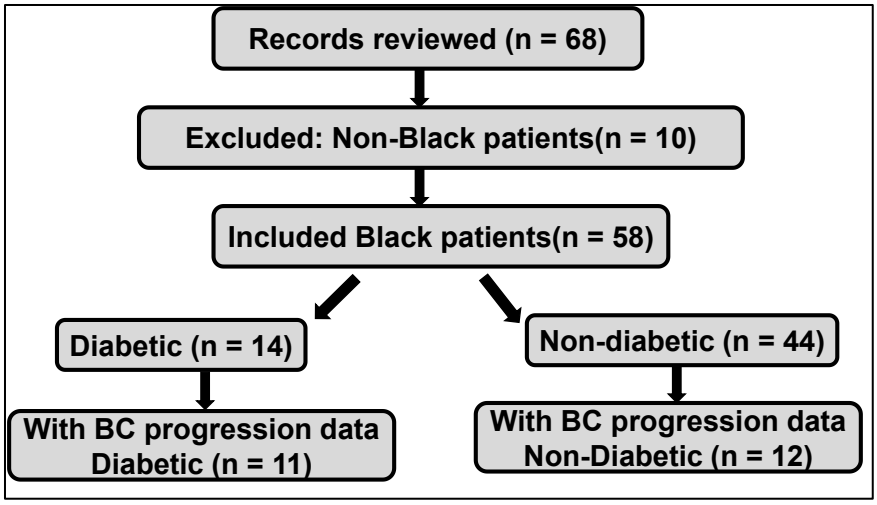

Figure 2. Flow Chart of Retrospective Study Design

DISCUSSION

Age is a well-established and non-modifiable risk factor for breast cancer and in the United States, only about 4% of BC cases occur in women under 40 years of age[23]. Our data showed that approximately 10% of Black women diagnosed with BC were aged 40 or younger, alluding to an earlier onset in our population. Additionally, the median age of BC diagnosis is consistently lower in Black women (60 years) compared to White women (64 years), underscoring potential racial disparities in disease biology and detection[23,24].

Menopause marks a critical physiological transition, with the median age in the US being 51 years, though this varies by race and ethnicity, for example, with studies indicating Black women tend to reach menopause at around 49 years of age[25]. Menopause itself is not a direct risk factor for BC, but the associated weight gain during this period significantly elevates BC risk[15,26], as the risk of BC increases by 12% with every 5-unit rise in BMI[28].

Obesity is a critical driver of insulin resistance (IR) and subsequent development of type 2 DM[32] by impairing glucose uptake in fat, muscle, and liver cells, resulting in persistent hyperglycemia. The cyclical relationship between obesity and IR raises an important question: which condition precipitates the other? Regardless, mounting evidence links DM to a 27% increased risk of BC[34], as DM has been shown to enhance aromatase expression and estrogen production, fueling the growth of estrogen receptor-positive (ER+) BC[36,37]. Epidemiologically, women with type 2 DM exhibit a 15% higher incidence of BC compared to non-diabetics[38]. In our cohort, 24% of Black women with BC had DM as a comorbidity; however, only 6.9% had DM before BC diagnosis, with the majority (17.2%) developing DM post-BC diagnosis. Notably, a modest increase in BC progression was observed among diabetics compared to non-diabetics, suggesting a contributory role of DM in disease advancement.

There is substantial evidence implicating adjuvant hormone therapy in the development of MS among BC patients[39,40]. Hormonal agents, including tamoxifen, a selective estrogen receptor modulator, and aromatase inhibitors such as anastrozole, letrozole, and exemestane, are cornerstone treatments for ER+ BC[41]. Our retrospective study revealed that over 70% of patients were prescribed hormone therapy, and strikingly, 70% of Black women on these therapies developed DM. Given that most of these women were already overweight or obese, this likely synergistically exacerbates insulin resistance and promotes BC progression, as reflected in our findings (**Figure 1**).

This study's findings carry critical clinical implications. The emergence of secondary diabetes in Black women undergoing hormone therapy for BC underscores the urgent need for vigilant metabolic monitoring and intervention. Overweight and obesity further compound the risk of insulin resistance and uncontrolled hyperglycemia, which may accelerate BC progression and complicate treatment outcomes. Regular assessment of glycated hemoglobin (HbA1c) alongside BC disease monitoring should be integrated into patient care.

This study represents preliminary pilot data exploring the metabolic consequences of endocrine therapy in Black women with breast cancer. Our findings suggest that majority of diabetes cases emerged following the initiation of hormonal therapy, indicating a potential treatment-related metabolic shift. Comparative analyses showed a higher rate of cancer

progression among diabetic patients (27%) compared to non-diabetic patients (8%), however, statistical testing did not yield significance (Fisher's exact p = 0.29), and adjusted logistic regression was limited by sample size and model instability.

This study has limitations, including a small sample size and retrospective design, which limit the ability to establish definitive causality between metabolic syndrome and BC progression. Additionally, the use of two separate electronic medical record systems introduced inconsistencies and potential data duplication. Clinician access to only one EMR system may have introduced information bias. Future prospective, larger-scale studies are warranted to further elucidate these complex relationships and to develop targeted interventions for Black women with BC. The median follow-up of 108 months supports the robustness of longitudinal observation; however, the study was underpowered (achieved power = 0.189) to detect definitive differences. These results are hypothesis-generating and underscore the need for larger, prospective studies to validate the observed trends and further investigate the role of endocrine therapy in diabetes onset and breast cancer progression in this vulnerable population.

We advocate for multidisciplinary strategies involving healthcare providers, community health workers, and pharmacists to support Black women with BC, particularly those receiving hormone therapy, in achieving optimal glycemic control and weight management. Such efforts will be pivotal to improving quality of life, reducing comorbidities, and potentially attenuating BC progression in this vulnerable population.

CONCLUSION

Our study highlights a concerning trend: a disproportionately higher percentage of younger Black women are being diagnosed with BC compared to the age-matched general population. A significant majority of these women were overweight or obese, reinforcing the role of obesity as a primary modifiable risk factor in the development of BC, more so than DM alone. Furthermore, our findings suggest a possible association between endocrine therapy and the onset of diabetes in Black women with breast cancer. However, given the retrospective design and the presence of confounding factors such as obesity and pre-existing metabolic risk, causality cannot be established. Further prospective studies are needed to clarify this relationship.

Given the well-documented role of DM in promoting BC progression through mechanisms involving chronic inflammation, hormonal dysregulation, and metabolic dysfunction, there is a critical need to proactively address this risk. This underscores the importance of comprehensive patient education focusing on lifestyle interventions, namely, glycemic control, regular physical activity, and a nutrient-rich diet, as integral components of BC management and survivorship, particularly in Black women.

To validate and expand upon these findings, there is a pressing need for large-scale, prospective, and randomized controlled studies that can further delineate the relationship between DM and BC progression. Specifically, future research should aim to clarify the role of hormone therapy in inducing secondary diabetes and explore targeted strategies for risk mitigation in vulnerable populations. Such efforts are vital to closing existing racial disparities and improving clinical outcomes in Black women with BC.

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